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TREATMENT OF DEAFNESS AND CONTIGUOUS NERVOUS DISORDERS WITH PROSTIGMINE

A PRELIMINARY CLINICAL REPORT

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Many interesting and informative reports on the use of prostigmine in the diagnosis and treatment of myasthenia gravis are being published. The effect of this drug on the nerves supplying the facial tissues has been fairly well studied. Yet it seems strange that the effect of the drug on the ear has not been noticed by those using the remedy in other conditions.

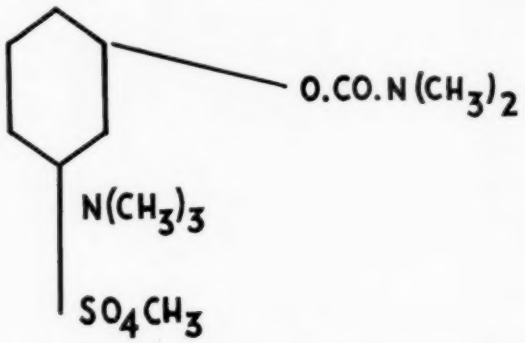
Our study of prostigmine began with a personal experiment by one of us (Dr. Rommel). In October 1936 he hypodermically injected 1 cc. of prostigmine prophylactic (1 : 4,000 solution) into himself in an experiment to learn something about the reactions of the tissues to this complex chemical. He found that it made his sense of hearing much more acute. A bedroom tickless clock, which makes no louder sound than a watch, became audible at a distance of 8 feet (245 cm.). This, he thought, is something deserving further study.

He then proceeded to use prostigmine prophylactic for hard of hearing patients. The first injections of this remedy for deficient hearing were given on Oct. 23, 1936, to an elderly woman (aged 60) and an elderly man (aged 76). At the next visit, four days later, both reported that they could hear better. At the end of ten days, without further injection, the improvement in hearing had ceased, and the condition had lapsed to the preinjection state, but Rommel had found that the remedy had acted for more than seven days. Injections were made thereafter at ten day intervals, at seven day intervals and then at three day intervals, and later prostigmine full strength, 1 : 2,000, was used. In April 1937 tablets of 15 mg. of prostigmine were given, one tablet to be taken on days when injection was not made. At first only one-half tablet was administered daily. The effort was being made to determine the proper frequency for the injections and for the use of the tablets.

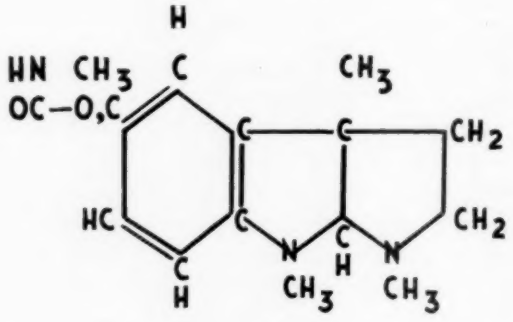
The hypodermic injections of prostigmine produced positive improvement in acuity of hearing. Rommel has not been able to determine any definite result from the use of the tablet in the treatment of deafness.

Prostigmine methylsulfate solution is put in 1 cc. ampules of 1 : 2,000 strength and 1 : 4,000 strength, and the bromide (instead of the methylsulfate) in tablets containing 15 mg. each. We used the 1 : 2,000 ampules after first using the 1 : 4,000 ampules. The best results were obtained with the stronger solution.

Prostigmine was synthesized in the Hoffmann-LaRoche laboratories. Chemically it is the dimethylcarbamic ester of 3-hydroxyphenyltrimethylammonium methylsulfate and has the following structural formula:



Physostigmine is an alkaloid obtained from the dried ripe seed of Physostigma venenosum Balfour. Its salicylate salt is official in the eleventh decennial revision of the Pharmacopeia of the United States; its molecular weight is 413.23, and its graphic formula is as follows:



It will be noted that prostigmine is a synthetic compound resembling physostigmine but differing from this alkaloid chemically by its less complicated structure and its greater stability. Solutions of prostigmine are stable. On the other hand, aqueous solutions of physostigmine salts oxidize under the influence of light, air and the alkali of glass vessels, acquiring a pink color, and then should not be employed.

Pharmacologically, in comparison with physostigmine, prostigmine is marked by a more pronounced action on peristalsis, a less pronounced miotic effect and the almost complete absence of cardiac by-effects.[1]

Schweitzer and Wright [2] studied, on cats under anesthesia induced by chloralose (a preparation of chloral hydrate and dextrose), the action of physostigmine and related substances and of acetylcholine on the central nervous system. The knee jerk was used as the typical somatic reflex. They found physostigmine (intravenous injection of 0.02 mg. per kilogram) greatly increases the knee jerk and general reflex excitability; occasionally there is an initial inhibition. Atropine sulfate (0.5 to 2.0 mg. per kilogram) commonly depresses the knee jerk, owing to a central action; it does not antagonize the excitatory action of physostigmine on reflex activity. Physostigmine influences the knee jerk by a direct action on the lower spinal centers; this may be "strychnine-like," facilitating transmission, or "ganglionic," increasing the excitability of anterior horn cells, or both. Prostigmine and Stedman's "meta" compound [3] depress the knee jerk, owing to a direct inhibitory action on the spinal cord; the inhibition is abolished by suitable doses of physostigmine. Prostigmine, acetylcholine and the "meta" compound can abolish convulsions induced by strychnine. Acetylcholine, 0.03 mg. per kilogram or over, usually depresses the knee jerk in unatropinized animals; the effects of small doses are annulled by atropine sulfate. Physostigmine increases the inhibitory effects of acetylcholine on the knee jerk, mainly because of the intensification of its central inhibitory action. The initial inhibitory action of physostigmine and the central inhibitory action of prostigmine and the "meta" compound are probably due to an anti-choline-esterase action on the central nervous system.

Schweitzer and Wright [4] stated that experiments show that prostigmine is anticonvulsant through a combined central and peripheral inhibitory action in varying proportions. Prostigmine exercises in the intact animal an antistrychnine action which is essentially identical with that produced by means of acetylcholine. The minimum dose of prostigmine necessary to inhibit the convulsions is usually 0.5 to 1.0 mg. The anticonvulsant action of the drug is independent of alterations in blood pressure or respiration.

Concerning physostigmine, the authors just mentioned stated:

> We have previously shown that eserine [physostigmine] has a dual action on the knee jerk which may possibly be attributed to two different components of the

1. Aeschlimann, J. A., and Reinert, M.: J. Pharmacol. & Exper. Therap. **43**:413, 1931. Berk, L.: Arch. f. exper. Path. u. Pharmakol. **168**:638, 1932.
2. Schweitzer, A., and Wright, S.: J. Physiol. **89**:165, 1937.
3. The methylcarbamic ester of metahydroxyphenyltrimethyl ammonium iodide.
4. Schweitzer, A., and Wright, S.: J. Physiol. **90**:310, 1937.

molecule: (*a*) it potentiates the inhibitory action on the central nervous system of subsequently injected acetylcholine because its urethane grouping exercises an inhibitory effect on cholinesterase, and (*b*) it has a strychnine-like action on the spinal cord increasing reflex excitability and producing convulsions. The grouping in the molecule responsible for action (*b*) is not yet known. We have previously recorded . . . that in some experiments eserine produces an initial inhibition of the knee jerk which may be due to inhibition of the cholinesterase and which is followed by the characteristic strychnine-like effect, i. e. the increase in reflex excitability.

In summarizing their studies, Schweitzer and Wright stated they had studied in cats the effects of intravenous injections of acetylcholine, doryl (carbaminoylcholine) and certain anti-choline-esterases (prostigmine, Stedman's "meta" compound and physostigmine) on strychnine convulsions. They found all these "autonomic" drugs, with the exception of physostigmine, diminish, abolish or delay the onset of strychnine convulsions. Physostigmine, which itself has a strychnine-like action on the spinal cord, does not influence the convulsions.

Myerson and others [5] called attention to the fact that Straub and Scholz,[6] studying both physostigmine and prostigmine, indicated that the inhibition of esterases is greater when prostigmine is used. Myerson and his co-workers employed prostigmine in their studies instead of physostigmine because it seemed to them prostigmine is more effective.

In November 1936 one of us (Dr. Rommel) proposed that we both use and study prostigmine in the treatment of deafness. When we had verified our findings further, as detailed later in this paper, we began to search for the reason. We felt that the muscles and nerves of the ear were probably associated with those of the face that are stimulated in cases of myasthenia.

Dr. William C. Pritchard, Professor of Histology and Embryology in Temple University School of Medicine, has given the following convincing data: [7]

> The branchiometric mass of the mandibular arch [part of the first arch] gives rise to the muscles of mastication (*temporal, masscter* and *pterygoid*), to the *mylohyoid, anterior belly of the digastric, tensor veli palatini* and the *tensor tympani.* These muscles are innervated by the trigeminal nerve [motor division of the trigeminal or fifth nerve].
>
> The branchiomeric mass of the second arch gives rise to the *stylohyoid,* the *posterior belly of the digastric, stapedius, platysma, occipitofrontalis* and the *muscles of expression;* these are innervated by the facial nerve.

5. Myerson, A.; Rinkel, M.; Loman, J., and Myerson, P.: J. Pharmacol. & Exper. Therap. **60**:296, 1937.

6. Straub, W., and Scholz, J.: Arch. f. exper. Path. u. Pharmakol. **182**:331, 1936.

7. Jordan, H. E., and Kindred, J. E.: A Textbook of Embryology, ed. 3, New York, D. Appleton-Century Company, Inc., 1937, p. 489.

Thus a common embryologic origin and a common innervation is found for the muscles of the ear and the muscles of the face that are so much paralyzed in myasthenia. If the latter are stimulated by prostigmine, of course, the former are.

Drs. A. M. Harvey and M. R. Whitehill, of Baltimore,[8] stated that Dr. Mary B. Walker[9] reported a striking improvement in a case of myasthenia gravis. After a hypodermic injection of 2.5 mg. of prostigmine, ptosis and ophthalmoplegia disappeared within five minutes.

Harvey and Whitehill reported the case of a Negro girl of 17, who had difficulty in mastication, ptosis, thick speech, regurgitation of water through the nose, transient diplopia and weakness of the arms. Examination disclosed bilateral ptosis, weakness of the right lateral rectus muscle, nystagmus in all directions, bilateral weakness of the muscles of the jaw, bilateral facial weakness and weakness of the arms, and gave the typical myasthenic electrical reaction. The patient was given hypodermically 1.5 mg. of prostigmine and 0.6 mg. of atropine sulfate. Within ten minutes she was able to open her eyes widely, and in twenty minutes the lips could be closed tightly. During the first several days in the hospital there were frequent attacks of severe dyspnea, during which the respiratory rate rose to 80 per minute. These attacks were promptly relieved by 1.5 mg. of prostigmine administered hypodermically.

Although dangers have been suggested by Boothby,[10] Minski and Stokes,[11] Hyland[12] and others, we do not feel that when a dose is given not oftener than every third day we have incurred any of those dangers. We found untoward symptoms in 2 women near middle life, 1 of them in menopause. Both had marked hypertrophy of the thyroid gland, and we immediately discontinued the treatment when it was reported that prostration and extreme nervousness followed every dose. No other patient has shown any untoward effects, although one of us (Dr. Davis) has given the drug to some for five months without interruption.

One of us (Dr. Davis) has studied 28 cases of *acute* blocking of the eustachian tube with tinnitus aurium, in most of which the condition was accompanied by marked deafness. Few of the patients have required more than five doses of prostigmine, even when the condition has lasted

8. Harvey, A. M., and Whitehill, M. R.: Prostigmin as an Aid in the Diagnosis of Myasthenia Gravis, J. A. M. A. **108**:1329 (April 17) 1937.

9. Walker, M. B.: Case Showing Effect of Prostigmin on Myasthenia Gravis, Proc. Roy. Soc. Med. **28**:759, 1935.

10. Boothby, W. H.: Treatment of Myasthenia Gravis, Canad. M. A. J. **34**: 272, 1936.

11. Minski, L., and Stokes, A. B.: Treatment of Myasthenia Gravis, Brit. M. J. **1**:1095, 1936.

12. Hyland, H. H.: Treatment of Myasthenia Gravis, Canad. M. A. J. **34**: 372, 1936.

a month. He accompanied the prostigmine with catheterization of the eustachian tube and massage. He feels that it is expecting too much to depend entirely on the prostigmine, although he has, with the consent of the patient, used it in 2 cases in which the condition was of one week's standing, without other medication. One of the 2 patients lost all noise in the head after three doses and the other after four. Two patients of this series had recurrences within three or four weeks, but both were cured by one treatment. They were all given 1 cc. doses of a 1:2,000 solution at intervals of every third to every fifth day.

One of us (Dr. Davis) has studied 29 cases of *chronic* deafness with tinnitus aurium, 20 of which are charted in the table. The other of us (Dr. Rommel) has added 4 cases to those listed in the table (21 to 24).

One of us (Dr. Davis) has used prostigmine successfully against trigeminal neuralgia. One patient remains free from pain after five months. Others under treatment are improving. This is a new treatment for this condition.

We here present a first report on the use of prostigmine in this wide and varied field. We cannot find in medical literature any statement that prostigmine has been found to improve or cure deafness. If others have observed it, we wish to add our studies.

The audiometer is now being standardized, after which it will be of great service in carefully recording the exact value of this drug in restoring hearing. However, this preliminary report is presented with the hope that the study of prostigmine in this most fertile and hopeful field will receive as much consideration as it has in the treatment of myasthenia gravis, in which the prognosis is sure to be discouraging.

SUMMARY AND CONCLUSIONS

1. Patients whose condition is acute usually get well (restoration of hearing) with five treatments or less.

2. Those whose condition is chronic require a longer time. It is advisable to continue the treatment indefinitely for chronic conditions. Progress is slow in many cases. We both have been tempted to give up the treatment for some patients, but improvement was brought about by persevering when hope seemed to have fled. Injections should be given two or three times a week for both acute and chronic conditions. When the condition was extreme positive results from the ingestion of prostigmine tablets could not be determined.

3. Prostigmine has real value against deafness and tinnitus aurium. It has great possibilities against trifacial and other neuralgias, which deserve further study.

4. It need not be used in such large amounts or so frequently that there is danger of reaching toxic dosage. Prostigmine should be studied until its value and its limitations in these fields are definitely determined.

Data in Twenty-Four Cases of Chronic Deafness with Tinnitus Aurium

Case Number and Patient	Age and Sex	Onset	Symptoms	Other Treatment	Number of Doses of Prostigmine Injected	Results
1 E. W.	35 M	5 yr. ago	Tinnitus bilateral; hearing, right ear 19/20, left ear 18/20	Inflation and massage	30 weekly	No change in tinnitus; hearing about normal
2 A. H.	48 F	2 yr. ago	Hearing, right ear 4/20, left ear 1/20; tinnitus bilateral	Inflation and massage	8 weekly, 29 triweekly	Hearing, right ear 10/20, left ear 6/20; no tinnitus right, slight left
3 L. H.	46 F	3 yr. ago	Constant never varying; tinnitus left; hearing, left ear 18/20, right ear normal	Inflation and massage	30 weekly	No change in tinnitus; slight improvement in hearing
4 A. K.	42 F	7 mo. ago	Tinnitus bilateral; hearing nearly normal; chronic sinusitis; hypertrophic thyroid	Inflation and massage	6 triweekly	Marked reaction due to thyroid condition; treatment discontinued
5 F. F.	53 F	5 yr. ago	Hearing, right ear 6/20, left ear 5/20; marked tinnitus; hypertrophic thyroid	Inflation and massage	3 weekly	Marked reaction due to thyroid condition; treatment discontinued
6 G. W. D.	43 M	10 yr. ago; symptoms marked	Hearing, right ear almost totally deaf, left ear 2/20; chronic pansinusitis, obstructing ridge left	Inflation and massage removal of ridge; Proetz displacement	28 weekly, 100 triweekly	Hearing in left ear 4 ft., no tinnitus; loud noises heard in right ear
7 N. C. D.	63 F	6 mo. ago; marked tinnitus	Right ear marked tinnitus, slight deafness; left ear tinnitus 3 weeks	Inflation and massage	16 weekly	Hearing about normal; no tinnitus
8 A. B.	24 M	Right ear deaf since childhood; left ear intermittently for 2 yr., growing worse for 1 yr.	Hearing, right ear 5/20, left ear 10/20	Inflation and massage	20 weekly (unable to continue)	6 mo. later hearing in right ear 15/20, left ear 18/20; no tinnitus
9 W. L. B.	42 M	8 mo. ago	Hearing, 15/20 bilateral	Inflation and massage; change of denture	7 at intervals	Came because of relapses; none after denture was changed
10 C. J. F.	18 M	5 yr. ago	Constantly increasing deafness; hearing, right ear 2/20, left ear 1/20; tinnitus bilateral; used hearing aid	Inflation and massage	8 weekly, 96 triweekly	Hearing, right ear 6/20; left ear 4/20; slight tinnitus left only; under treatment
11 C. W. A.	69 M	1 yr. ago	Fulness and deafness; hearing, left ear 12/20, right ear normal	Inflation and massage	51 triweekly	Ear felt clear after 10 doses; hearing, 19/20
12 W. L. R.	51 M	3 yr. ago	Pansinusitus; right ear discharging at times; hearing, right ear 5/20, left ear 18/20; marked vertigo	Inflation and massage; Proetz displacement	10 weekly, 60 triweekly, irregularly given when no symptoms appeared	No vertigo for 5 mo.; hearing, right ear 10/20, left ear 20/20
13 H. B.	32 F	6 yr. ago	Gradually increasing deafness; hearing, right ear 4/20, left ear 8/20; bilateral tinnitus	Inflation and massage	(Treated before without much improvement); 13 weekly, 24 triweekly	Treatment discontinued 5/18/37; no buzzing; hearing about 25% better; returned 2/28/38, having relapsed to about original condition; no treatment for 2 wk.; no tests but improvement claimed

Data in Twenty-Four Cases of Chronic Deafness with Tinnitus Aurium—Continued

Case Number and Patient	Age and Sex	Onset	Symptoms	Other Treatment	Number of Doses of Prostigmine Injected	Results
14 J. G. M.	38 M	15 yr. ago	First 10 years gradually increasing deafness; hearing, right ear 3/20, left ear 1/20; no tinnitus	Inflation and massage; Proetz displacement	51 triweekly	Hearing, left ear 4/20, right ear 2/20
15 G. L.	42 M	7 yr. ago	Gradually increasing deafness, first left; hearing, 2/20 bilateral; no tinnitus	Inflation and massage	81 triweekly	Hearing about double
16 H. H.	49 F	7 yr. ago	Hearing, right ear 1/20, left ear 3/20; congenital syphilis	Inflation and massage	36 weekly irregularly	Hearing slightly improved, variable
17 R. C.	59 M	1 yr. ago; symptoms marked	Hearing, right ear 14/20, left ear 3/20; tinnitus bilateral	Inflation and massage	36 weekly	Hearing, right ear, 18/20, left ear 12/20; mild tinnitus left only; under treatment
18 F. E. L.	47 F	More than 20 yr. ago	Hearing, right ear no conversation, left ear 1/20	Inflation and massage; bilateral mastoidectomy 6 yr. ago	108 triweekly	Frequent relapses; no conversation still heard with right ear; hearing, left ear 3/20
19 M. M.	29 F	3 yr. ago	Constant tinnitus right; intermittent tube blocking right	Tonsillectomy 1 yr. ago	51	Ear clear after 10 injections; tinnitus intermittent and faint; still under treatment
20 S. A.	60 M	3½ yr. ago	Dulness and mild tinnitus; hearing, right ear 10/20, left ear 12/20; chronic pansinusitis	Inflation* and massage until 6 mo. ago; Proetz displacement	51 triweekly without other treatment	Hearing, right ear 14/20, left ear 16/20 at times; rarely feels dull, but hearing varies
21 L. P.	79 F	In childhood	Absolute deafness in right ear; used sonotone for bone conduction on left mastoid; required loud speaking to hear	None	8	Left ear excellent (discontinued sonotone); hears ordinary tones and conversation; right ear, no improvement
22 H. S.	48 M	1918	Hears only loud talking	Nasal treatment; inflation of eustachian tubes	4	Excellent
23 W. P. G.	46 M	1924	Felt as if something was in left ear	Nasal treatment; inflation of eustachian tubes	11	Improved satisfactorily
24 A. W.	68 F	1929	Gradual loss of hearing	Nasal treatment; inflation of eustachian tubes	33	Great improvement; still undergoing treatment

* We catheterize eustachian tubes once weekly and instil vapor by the method devised by Dr. Walter A. Wells (Laryngoscope **42**: 779, 1932).

In all cases in which prostigmine is used we feel that all causative factors, such as sinusitis, eustachian tubal catarrh and nasal obstructions, must be treated and as far as possible removed, in order to permit cure and prevent relapses. One should not rush the treatment too fast in any case.

PHYSIOLOGY OF THE VESTIBULAR LABYRINTH

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As a result of a recent experience with spastic torticollis associated with rather striking phenomena that would indicate a relation of torticollis to the vestibular apparatus, I present this monograph as a companion paper to the case report "Torticollis Spastica: Suggested Etiologic Relation to the Vestibular Apparatus: Report of a Case."

It is felt that a more or less extended review of vestibular physiology is in order before one attempts to understand the pathologic functions to which the apparatus when defective gives rise and their possible role in the development of torticollis spastica.

The presentation is given according to the following outline:

I. Physiology
   A. Experimental Observations on Animals
   B. Observations on Man

II. General Comment

III. Special Problems
   A. Physical Nature of the Inner Ear
   B. Role of the Cerebellum

The works of Camis, McNally, Ruttin, Jones, Meltzer, Maxwell and de Kleijn were consulted freely, and many of the references to foreign works were recorded from these authors.

I. PHYSIOLOGY

EXPERIMENTAL OBSERVATIONS ON ANIMALS

Semicircular Canals.—Flourens [1] was the first to describe nystagmus following injury to the semicircular canals in pigeons. He found that injury to a canal causes nystagmus in the plane of that canal. This became known as Flourens' law.

From the Department of Surgery, Neuro-Surgical Service, College of Medicine, State University of Iowa.

1. Flourens, P.: Recherches expérimentales sur les propriétés et les fonctions du système nerveux, dans les animaux vertébrés, ed. 2, Paris, J.-B. Bailliére, 1842.

Breuer [2] and Ewald [3] confirmed Flourens' law and showed, in addition, that the direction of flow of the endolymph in a single canal controls the direction of the nystagmus. If the endolymph in the right horizontal canal is forced toward the ampulla, the eyes move to the left. If the direction of the endolymph is away from the ampulla, the eyes move to the right. From these experiments Ewald's first law was evolved; viz., a horizontal canal is maximally stimulated by movement of the endolymph toward its ampulla, and each vertical semicircular canal is maximally stimulated by movement of the endolymph away from the ampulla. Ewald showed that this law applied to the pigeon; de Kleijn demonstrated it for the rabbit, and Ruttin stated that it is applicable to man.[4] Ruttin pointed out that after complete destruction of one labyrinth in man responses are obtained by turning in either direction but they are unequal. After some time such responses approach equality. The law does not hold, however, throughout the animal kingdom, as shown by Maxwell [5] for the dogfish and by McNally [4] for the frog. The latter when turned to the side on which the ampulla has been destroyed makes no response, but if the turning is stopped suddenly the animal turns in the direction to which it was originally being turned. If it is turned to the normal side, it responds by moving around in the opposite direction. Now, if the table is stopped suddenly the frog remains stationary. It is postulated, therefore, that the horizontal canal of the frog is stimulated by flow of endolymph in only one direction.

Ewald derived a second law from his experiments: that when any canal is maximally stimulated nystagmus is invoked to the ipsilateral side, whereas minimal stimulation causes nystagmus to the opposite side. (Nystagmus is arbitrarily spoken of as being toward the quick component.)

These laws of Flourens and Ewald have remained almost universally accepted. Tait and McNally,[6] in a number of well controlled experi-

2. Breuer, J.: Neue Versuche an den Ohrbogengängen, Arch. f. d. ges. Physiol. **44**:135, 1889.

3. Ewald, J. R.: Physiologische Untersuchungen über das Endorgan des Nervus octavus, Wiesbaden, J. F. Bergmann, 1892.

4. McNally, W. J.: Physiology of the Cochlea with Some Remarks About Clinical Tests for Hearing, Ann. Otol., Rhin. & Laryng. **38**:1163, 1929; Physiology of the Saccule and the Saccus Endo-lymphaticus, ibid. **38**:1180, 1929; Physiology of the Utricle and Its Association with Posture, ibid. **39**:248, 1930; Physiology of the Semicircular Canals, ibid. **39**:262, 1930; Some Clinical Applications of the Physiology of the Internal Ear, ibid. **39**:276, 1930.

5. Maxwell, S. S.: Labyrinth and Equilibrium, Philadelphia, J. B. Lippincott Company, 1923.

6. Tait, J., and McNally, W. J.: An Analysis of the Limb Responses to Semicircular Canal Stimulation in the Frog, Ann. Otol., Rhin. & Laryng. **38**:1121, 1929.

ments on frogs, confirmed the theory that the semicircular canals are responsible for responses obtained during angular acceleration and sudden changes in posture. McNally and his co-workers showed that if the nerves from the ampullae are cut or cauterized individually the frog will fail properly to adjust his posture during movement in the plane of the destroyed canal. Thus, if a left anterior vertical canal has been destroyed, the frog when jumping turns to the left and tends to land on the left shoulder with an inadequate thrust of the left arm. If both anterior vertical canals have been destroyed, the frog tumbles forward. If both posterior vertical canals have been destroyed, the frog tumbles backward. If all six canals have been destroyed, it is loath to jump and lands ungracefully with a tendency to oscillate back and forth. All such inadequate responses are manifest only during the movement. If the utricle is left intact, the animal will subsequently assume a relatively normal posture. A normal frog if placed in the center of a turn table, will turn in the direction opposite to the motion of the table. If then the canals on one side are destroyed, the animal will make no response when turned to that side but will make a normal response when turned to the normal side. If all six canals are destroyed, the animal will respond in neither direction.

Utricle.—The utricle plays an important role in governing posture and righting reflexes. It is an organ of static equilibrium. The evidence to date indicates that one position (supine for the cat) is associated with increased tone of the extensor muscles in all four extremities, whereas the opposite position is associated with flexion of the extremities.[7] One utricle seems to govern all four extremities and the muscles of the opposite side of the neck. The function of the utricular macula can be eliminated in guinea pigs by centrifuging.[8] Animals prepared in this way lose the tonic postural labyrinthine responses in the musculature of the body but retain the responses attributed to the semicircular canals.

There is some controversy concerning whether pulling or pushing of the plate of the macula on the hair cells constitutes the maximum stimulus. De Kleijn and Magnus showed that a decerebrate cat has maximum tonus of the extensor muscles in all four extremities when in the supine position, i. e., when the otoliths hang from the maculas. Conversely, the tonus is minimum when the otoliths rest on the maculas. These being the experimental facts concerning the relation of the otoliths to the tone of the extensor muscles, it would seem relatively unimportant

7. de Kleijn, A., and Magnus, R.: Ueber die Funktion der Otolithen: I Otolithenstand bei den tonischen Labyrinthreflexen, Arch. d. ges. Physiol. **186**: 6, 1921; II. Isolierte Otolithenausschaltung bei Meerschweinchen, ibid. **186**:61, 1921.

8. Wittmaack, K.: Ueber Veränderungen im inneren Ohre nach Rotationen, Verhandl. d. deutsch. otol. Gesellsch. **18**:150, 1909.

whether one regards the hanging otolith as the maximum or the minimum stimulus to the organ per se.

It has been difficult for me to understand why the hanging otolith, or supine position, is associated with maximum tonus of the extensor muscles. One would expect the tonus to be greatest when the animal is in the normal upright position, to counteract gravitational force. This will be referred to again in the discussion of the caloric test under "Special Problems."

The postural righting reactions involve the integration of stimuli from several sources, more particularly from the labyrinth and the tonic reflexes of the neck. If a decerebrate cat with labyrinths intact is rotated on a horizontal anteroposterior axis, the head will return to the original position with respect to the ground and maintain this position. The twist of the neck so produced is followed by reflex righting of the body.[9] When the head has righted itself, corrective stimuli no longer come from the labyrinths, and the righting of the body must then be purely a result of the tonic reflexes of the neck. If the head were splinted to the body, the labyrinthine reflexes would govern per se the entire body, and righting would be effected by them rather than by the disassociated mechanism which operates when the head is free to turn. In the Crustacea and lizards the undisassociated mechanism must obtain.

Stirling [10] pointed out that when the head of a pigeon is sutured to the chest the bird shows a marked loss of equilibrium.

The classic and ingenious experiment of Kreidl [11] (1893), in which the otoliths of the crayfish become replaced by iron filings, clearly demonstrates by the use of a magnet the function of this organ.

The otolithic organ is responsible also for certain compensatory positions of the eye.[12] The reflexes are best studied in animals with laterally placed eyes, In animals that have overlapping visual fields, narcosis is necessary for the study of these compensatory positions of the eye. If the head is fixed to the body and the body rotated down and to the right, the right eye moves up and the left eye down. In general, the tendency seems to be toward a fixation of the visual fields. The compensation, however, falls a little short of being complete (about 20 per cent).[13] The reflexes of the neck are associated with compen-

9. Magnus, R.: Physiology of Posture, Lancet **2**:531 and 585, 1926.

10. Stirling, W.: Functions of the Semicircular Canals, J. Anat. & Physiol. **8**:400, 1874.

11. Kreidl, cited by Bayliss,[86] p. 527.

12. Throughout the paper I shall refer to deviations of the eye that are governed by the otolithic organ as compensatory positions and to those that are governed by the canals as compensatory movements.

13. Bárány, cited by McNally.[4] Magnus and de Kleijn, cited by McNally.[4]

satory positions of the eye, and the two mechanisms together control a perfect fixation of the visual fields. The influence of the neck on compensatory positions or rolling of the eye is not lost, as is the influence of the utricle, after destruction of the labyrinths.

In well controlled experiments on the frog, McNally [14] demonstrated the influence of the utricle in maintaining posture when the animal is placed on a tilting plane or subjected to centrifugal force. These postural reactions are preserved after destruction of all six semicircular canals. A most interesting observation is brought out; viz., if the frog's body is attached to a cork platform, which is then raised so that the animal's limbs are unsupported, no changes in the tone of the extremities are manifested after the animal is tilted or when it is subjected to centrifugal force. This would indicate that the labyrinth and the proprioceptors must function as an integrated unit; i. e., the labyrinth does not bring to bear its influence on tone, so to speak, unless the animal is in kinesthetic contact with the environment against which it is to maintain balance.

Versteegh,[15] by destroying only the nerve from a utricular macula of the rabbit, showed that the utricle is the seat of the labyrinthine head-righting reflexes, the labyrinthine compensatory positions of the eye and the labyrinthine tonic influence on the musculature of the body and neck. The latter causes a turning of the head to the side of the utricular injury.

That the otolithic organ is not responsible for nystagmus has been demonstrated in a number of ways. One ingenious method used by Magnus and de Kleijn (1923) [16] involved the progressive cocainization of the labyrinth. There is evidence to show that the utricle and saccule become paralyzed without irritation first. This is later followed by deadening of the canals. Nystagmus does not make its appearance until the canals become paralyzed. The horizontal canal is the first to become paralyzed.

Saccule.—The evidence to date seems to favor the conclusion that the saccular macula has no vestibular or equilibrial function in the mammal or in the lower animals studied. Laudenbach (1899), by washing away the saccular otolith in the frog, McNally and Tait (1925), by sectioning the nerve to the saccular macula on one or both sides, and Versteegh (1928), by experimental ablation in the rabbit, demonstrated that all known labyrinthine functions attributable to the otolithic mechanism are functions of the utricle and not of the saccule.

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14. McNally, W. J.: Experiments on the Utricle, Laryngoscope **44**:50, 1934.

15. Versteegh, cited by McNally.[4]

16. Magnus and de Kleijn, cited by Camis.[33]

De Kleijn and Versteegh [17] at first thought that the labyrinthine righting reflexes and compensatory positions of the eye were governed by the saccular macula and that the tonic labyrinthine reflexes on the musculature of the body were governed by the utricular macula. They cited Quix [18] as believing that torsion of the head, as well as abduction and adduction of the limbs, is due to the loss of one saccule. However, after developing a technic for removing the saccules bilaterally in rabbits, de Kleijn and Versteegh found no disturbance in any of the known functions of the labyrinth and therefore stated that their previous theories and that of Quix were impossible.

Views to the contrary have been expressed by Magnus,[9] who stated that the utricles govern symmetric righting reflexes and the saccules assymetric ones. His reasoning was that the utricular maculas lie in one plane, and the saccular maculas do not, so that after unilateral labyrinthectomy, the asymmetric position of the head on the body (being a permanent posture) cannot be due to the utricle.

Benjamins and Huizinga [19] after selective operations on labyrinths of pigeons concluded that for animals with laterally placed eyes, at least, rolling of the eyes is governed by the saccules.

Effects of Partial or Complete Labyrinthectomy.—The effects of partial and of complete labyrinthectomy have been studied in a number of different animals—the pigeon, frog, guinea pig, rabbit, cat, dog, monkey and, in the course of clinical work, the human being.

Except, for obvious reasons, in man, responses demonstrated in different animals have been basically similar. There have been minor differences in the relation of nystagmus and compensatory positions of the eye, which can be attributed to the anatomic position of the eyes—whether laterally placed or placed so as to cause overlapping of the visual fields. Nystagmus of the head is a striking reaction in the pigeon while not so prominent in the frog; here again the difference is attributable to anatomic differences. Bilateral representation of the canals appears to be absent in the frog but present in the cat. On the whole, however, certain fundamental phenomena are exhibited by all animals studied.

Pigeon: Destruction of a single canal causes nystagmus of the head in the plane of the canal, and destruction of the symmetrically corresponding canal greatly augments the response. Any stimulus to the bird emphasizes the response after it has quieted.

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17. de Kleijn, A., and Versteegh, C.: Some Remarks on the Present Position of the Physiology of the Labyrinth, J. Laryng. & Otol. **42**:649, 1927.

18. Quix, F. H.: The Function of the Vestibular Organ, J. Laryng. & Otol. **40**:425, 1925.

19. Benjamins, C. E., and Huizinga, E.: Die Raddrehung wird bei Tauben von den Sacculusotolithen ausgelöst, Ztschr. f. Hals-, Nasen- u. Ohrenh. **15**:228, 1926; abstracted, J. Laryng. & Otol. **42**:404, 1927.

Destruction of all canals on one side causes oscillation of the head, a tendency to fall and to circle to that side and limping on the same side. A striking response is rotation of the head. Pictures, taken from Ewald, of pigeons in such an attitude have been presented by Camis.[20] These pigeons are able to drink and peck grain.

Pigeons with both labyrinths destroyed present oscillation of the head, staggering, zig-zag gait, inability to fly and inability to drink or to peck grain.

Frog: McNally's work has been outlined previously. Though he used different operative methods, the results are in agreement with those of Ewald and also of Camis. After unilateral labyrinthectomy, the head is rotated so that the side operated on is dependent. The limbs of that side are adducted and flexed. Those of the opposite side are abducted and somewhat extended.

Guinea Pig: After unilateral extirpation, the animal's head is rotated with the side operated on dependent, and there is nystagmus of the head. The eyes are deviated to the side operated on, a response which is accompanied by nystagmus. These responses disappear in several days, but thereafter if the animal is held suspended with the head downward there is rotation of the head and axial rotation of the body, with the forelimb on the side not operated on extended. This reaction appears to be permanent.

Rabbit: Winkler, Camis and Magnus expressed agreement on the following points: After unilateral labyrinthectomy, there are ocular nystagmus and rolling movements. The latter are at first spontaneous, but later can be evoked by stimulation.

More permanent phenomena are spiral rotation of the body and rotation of the head with the side operated on resting dependent. The ipsilateral forelimb is flexed and adducted, while the opposite is extended and abducted. Deviation of the eyes is also of long duration. On the side operated on the eye is deviated downward and inward, while the opposite eye is deviated upward and outward.

McNally[4] stated that after unilateral labyrinthectomy there is marked turning of the head to the side operated on. (He did not mention the animal used but it was probably the rabbit.) There is loss of compensatory movements following turning of the head to the side operated on; viz., after right-sided labyrinthectomy, the right eye is turned down and the left up, which is contrary to what happens in the normal animal. There is loss of labyrinthine head-righting reflexes on turning the head to the side operated on. There is increased tone in

20. Camis,[33] p. 51.

the extensor and abductor muscles of the homolateral limbs [21] and increased flexion of the contralateral limbs. It was proposed by Magnus and de Kleijn that the asymmetry of the limbs is really a reflex of the neck because if the head is brought into symmetry with the rest of the body the asymmetry of the limbs disappears.

Cat and Dog: The responses after unilateral labyrinthectomy are similar in these animals. The cat (Kreidl, Camis and Magnus) presents mild and brief deviated (compensatory) positions of the eye, while this phenomenon is more marked in the dog. The head is rotated with the side operated on dependent, and the reaction tends to be more permanent in the dog. The cat does not exhibit rolling movements, while the dog does, as a temporary phenomenon. Both present temporary ocular nystagmus, horizontal and rotary, with the rapid component toward the side not operated on.

Monkey: Magnus (1922)[22] recorded the immediate effects of unilateral labyrinthectomy as deviation of the eyes and horizontal nystagmus, lasting two or three days, rolling movements when the animal attempts to walk about, hypotonicity of the limbs on the side operated on, rotation and flexion of the head toward that side and nystagmus of the head. The rotation and flexion of the neck are permanent.

Northington and Barrera [23] studied the responses of monkeys after extirpation of the labyrinth as well as after section of the eighth nerve, unilateral and bilateral. The results were the same whether the labyrinth in question was destroyed or the eighth nerve sectioned. The following is a representative protocol:

> After destruction of the left ear, the occiput was turned toward the left shoulder and the chin rotated to the right. There was spontaneous nystagmus to the right, which did not vary with changes in the position of the head. After twelve hours the nystagmus gradually disappeared. On recovering from the anesthetic, the monkey showed some jerkiness of the body and head. A fine shaking of the head seemed to be synchronous with the nystagmus. The left extremities felt more flaccid and the right extremities were extended, though straightening the head when the animal was supine tended to equalize the tone. The animal chose to lie on the left side. It showed ipsilateral lateropulsion and falling, spiraling when climbing a bar, ipsilateral deviation when jumping and loss of orientation in water.
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> The caloric stimulation (cold) in the right ear produced normal responses, which were characteristic with the chin up and with the chin down. Rotation

21. This is contrary to other reports and is probably a typographic error. The contralateral limbs are extended and abducted.

22. Magnus, R.: Körperstellung und Labyrinthreflexe beim Affen, Arch. f. d. ges. Physiol. **193**:396, 1922.

23. Northington, P., and Barrera, S. E.: Effects of Unilateral and Bilateral Labyrinthectomy and Intracranial Section of the Eighth Nerve, Arch. Neurol. & Psychiat. **32**:51 (July) 1934.

gave normal but diminished responses. The posture of the head was reversed by section of the ipsilateral cervical nerves.[24]

The other ear was destroyed two months later. The same reactions occurred in the opposite direction but were less marked. The nystagmus, however, was vertical and feeble.

The caloric and turning tests produced no responses. In the galvanic test, the cathode on the ear newly operated on produced a good normal response with 10 milliamperes, while the anode produced no reaction even with 25 milliamperes.

The monkey shuffled and slid with a broad gait. It was unable to adjust the body and limbs to rapid changes in the position of the head, and there was marked disability in jumping. It assumed bizarre postures, in which there was a tendency to dorsiflex the head.

OBSERVATIONS ON MAN

Pathologic Situations.—Striking pathologic situations in man are tumor of the eighth nerve, Ménière's disease and labyrinthitis.

Though it is precarious to draw rigorous physiologic conclusions when a massive lesion such as a tumor exists, it is nevertheless interesting that a deviation of the head to the affected side often occurs in cases of tumor of the eighth nerve.

There seems to be sufficient evidence that Ménière's syndrome is a phenomenon of the end organ. It is associated with progressive deafness and may be abolished by section of the ipsilateral vestibular nerve.[25] Analysis of the seizures leads one to feel that the *locus operandi* is in the semicircular canal system. Certainly persons without vestibules are incapable of experiencing the subjective dizziness that characterizes Ménière's syndrome.

Labyrinthitis, within the first few days, causes nystagmus to the homolateral side.

Ruttin pointed out that injury to the human labyrinth causing a release of endolymph produces the same reaction and signs that follow extirpation of the labyrinth.

Effects of Section of a Vestibular Nerve.—Dandy [26] reported the following sequelae of section of a vestibular nerve:

Diplopia: This occurred in 4 cases and persisted only a few days. No weakness of the extraocular muscles could be demonstrated. (Nearly

24. McNally [4] concluded from experimental work on the rabbit that impulses from the intact labyrinth cross to the cervical nerves of the opposite side (the first five cervical nerves and the accessory nerve) and that the connection is made in the cord rather than in the medulla. He stated that the cervical nerves on the intact side oppose and may partly mask the action of the nerves on the side of the labyrinthectomy.

25. Dandy, W. E.: Ménière's Disease: Its Diagnosis and Method of Treatment, Arch. Surg. **16**:1127 (June) 1928; in Lewis, D.: Practice of Surgery, Hagerstown, Md., W. F. Prior Company, Inc., 1932, vol. 12, p. 207.

26. Dandy, W. E.: Ménière's Disease: Symptoms, Objective Findings and Treatment in Forty-Two Cases, Arch. Otolaryng. **20**:1 (July) 1934.

all the patients of Cairns and Brain had diplopia. I feel that this phenomenon may be related to the asymmetric ocular reflexes demonstrated in animals.)

Transient Nystagmus: This occurred with considerable frequency but also disappeared within a few days.

Transient Vertigo: This usually began on the day of the operation, reached its peak within forty-eight hours, then declined steadily and had practically disappeared at the end of a week or ten days. Nausea and vomiting were frequently associated with the dizziness. Dizziness induced by turning the head persisted longer, occasionally for a year or more, and in a single patient for three years. One patient made it clear that turning the head provoked a disturbance of balance but no dizziness.[27] Occasionally the vertigo may be entirely absent. (I feel that the degree to which postoperative signs are manifested depends on the degree to which the end organ has become destroyed during the progress of the disease. The same holds true for tumor of the eighth nerve, after the removal of which such signs may be feeble.)

I have had the fortune to study postoperatively 2 of Dr. Dandy's patients in each of whom bilateral section of the vestibular nerves had been done for Ménière's syndrome, the sections being separated by a long interval of time. The immediate reactions observed in animals, such as nystagmus and deviation of the head, were not present in Dandy's patients at the time I studied them. (One patient I studied personally about ten days after the operation. The facts concerning the other patient were carefully elicited by Dr. Verbeek in Dandy's clinic.) The phenomena brought out here appear to last a long time.

If the patient stands with eyes closed and one rotates his head to the side, so far from being subjectively aware that his head has turned, he feels that his feet and body have rotated in the opposite direction. This sensation is undoubtedly due to the disassociation of proprioceptive information from the muscles of the neck and afferent stimuli from the vestibule. His proprioceptive sense of position informs him that the head has changed position in relation to the body, but the absence of vestibular stimuli can but inform him that his head did not move, and he thus receives the alternative subjective impression. He responds to all the usual neurologic tests normally, and his posture in standing and walking appears normal, but if he suddenly turns his head to the side he becomes ataxic. Here again, the afferent stimuli from the ves-

27. A patient (K. S.) in whom I had totally removed a perineural fibroblastoma of the root of the fifth nerve and had destroyed the vestibular portion of the eighth nerve complained for a year of imbalance, more pronounced on sudden changes in position, and strictly declared that she did not experience dizziness or vertigo. The sensation was progressively but slowly diminishing, and rotation tests a year after operation indicated that full compensation and adaptation to the remaining labyrinth had not yet occurred.

tibule, formerly depended on, fail to contribute their tonal values, and failure to contribute the psychic adjunct is confusing.

Dandy [28] stated that a patient of his could not saw along a straight line. This disability would be expected and is a striking demonstration of the labyrinthine-ocular reflexes and the function of the labyrinth in maintaining fixed vision though the head may be moving about in all directions. It is common experience that though one may be riding in a vehicle over a rough road one can fix one's vision steadily on a given point in the landscape.

Physiologic Tests.—The Bárány tests are well known. For details the reader is referred to Jones and Fisher.[29] In the physiologic tests on man four reactions are significant, nystagmus, vertigo, past pointing and falling.

Rotation Test: In rotation the character of the nystagmus is determined by the position of the head during turning. After the turning is stopped, the character of the nystagmus is maintained regardless of the position in which the head is placed. During rotation, the nystagmus ("with" nystagmus) is in the direction of turning. When the turning is stopped, the nystagmus ("after" nystagmus) is in the opposite direction.

As Bárány stated, no matter what the position of the head is in turning, the nystagmus will be parallel to the ground. With the head upright (30 degrees forward) the nystagmus is horizontal; with the head 60 degrees backward or 120 degrees forward, rotary, and with the head inclined to the shoulder, vertical. The reader is referred to Jones and Fisher [30] for the protocol of a normal subject in regard to time of onset, duration of nystagmus and magnitude of past pointing.

The following responses to rotation are taken from Jones and Fisher,[29] and are those which occur when turning is stopped: [31]

NYSTAGMUS

Turning to the right with the head upright produces horizontal nystagmus to the left.

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28. Dandy, W. E.: The Treatment of Bilateral Ménière's Disease and Pseudo-Ménière's Disease, Tr. Am. Neurol. A. **61**:128, 1935.

29. Jones, I. H., and Fisher, L.: Equilibrium and Vertigo, Philadelphia, J. B. Lippincott Company, 1918.

30. Jones and Fisher,[29] p. 251.

31. The degree to which the head is placed forward or backward is sometimes denoted by different figures. To be accurate, the horizontal canal is actually horizontal when the head is 30 degrees forward, and this should be considered the upright position. When displacements are reckoned from this as the upright, then the head is said to be back 90 degrees and forward 90 degrees. If displacements are reckoned from the mathematical vertical, the head is said to be back 60 degrees and forward 120 degrees. The forward and backward positions are identical in both cases.

Turning to the left with the head upright produces horizontal nystagmus to the right.

Turning to the right with the head 60 degrees backward produces rotary nystagmus to the right.[32]

Turning to the left with the head 60 degrees backward produces rotary nystagmus to the left.[32]

Turning to the right with the head 90 degrees forward produces rotary nystagmus to the left.

Turning to the left with the head 90 degrees forward produces rotary nystagmus to the right.

Turning to the right with the head inclined toward the right shoulder produces vertical nystagmus downward.

Turning to the left with the head inclined toward the right shoulder produces vertical nystagmus upward.

Turning to the right with the head inclined toward the left shoulder produces vertical nystagmus upward.

Turning to the left with the head inclined toward the left shoulder produces vertical nystagmus downward.

VERTIGO

Turning to the right with the head upright produces a sensation of turning to the left in the horizontal plane.

Turning to the left with the head upright produces a sensation of turning to the right in the horizontal plane.

Turning to the right with the head forward 120 degrees produces: (*a*) with the head kept forward, a sensation of turning to the left in the horizontal plane; (*b*) with the head brought upright, a sensation of falling to the left in the frontal plane.

Turning to the left with the head forward 120 degrees produces: (*a*) with the head kept forward, a sensation of turning to the right in the horizontal plane; (*b*) with the head brought upright, a sensation of falling to the right in the frontal plane.

Turning to the right with the head back 60 degrees produces: (*a*) with the head kept backward, a sensation of turning to the left in the horizontal plane; (*b*) with the head brought upright, a sensation of falling to the right in the frontal plane.

Turning to the left with the head back 60 degrees produces: (*a*) with the head kept backward, a sensation of turning to the right in the horizontal plane; (*b*) with the head brought upright, a sensation of falling to the left in the frontal plane.

PAST POINTING

Turning to the right with the head upright produces past pointing to the right.

Turning to the left with the head upright produces past pointing to the left.

Turning to the right with the head back 60 degrees produces past pointing to the right.

Turning to the left with the head back 60 degrees produces past pointing to the left.

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32. I have taken the liberty of making a correction in these two statements. Jones recorded that turning to the right produces nystagmus to the left and turning to the left nystagmus to the right.

Turning to the right with the head forward 120 degrees produces past pointing to the right.

Turning to the left with the head forward 120 degrees produces past pointing to the left.

Turning to the right with the head inclined toward the right shoulder and then bringing the head upright produces past pointing upward.

Turning to the left with the head inclined toward the right shoulder and then bringing the head upright produces past pointing downward.

Turning to the right with the head inclined toward the left shoulder and then bringing the head upright produces past pointing downward.

Turning to the left with the head inclined toward the left shoulder and then bringing the head upright produces past pointing upward.

FALLING

Turning to the right with the head forward 120 degrees and the subject sitting upright produces falling to the right.

Turning to the left with the head forward 120 degrees and the subject sitting upright produces falling to the left.

Turning to the right with the head back 60 degrees and the subject sitting upright produces falling to the left.

Turning to the left with the head back 60 degrees and the subject sitting upright produces falling to the right.

Turning to the right with the head inclined toward the right shoulder and bringing the head upright produces falling backward.

Turning to the left with the head inclined toward the right shoulder and bringing the head upright produces falling forward.

Turning to the right with the head inclined toward the left shoulder and bringing the head upright produces falling forward.

Turning to the left with the head inclined toward the left shoulder and bringing the head upright produces falling backward.

Caloric Test: The test consists of douching with cold water (68 F.) or warm water (112 F.) against the ear drum. The routine management was outlined by Jones and Fisher.[29]

The following responses have been listed from Jones and Fisher:

NYSTAGMUS

Douching the right ear with the head upright and the water at 68 F. produces rotary nystagmus to the left.

Douching the left ear with the head upright and the water at 68 F. produces rotary nystagmus to the right.

Douching the right ear with the head 60 degrees backward and the water at 68 F. produces horizontal nystagmus to the left.

Douching the left ear with the head 60 degrees backward and the water at 68 F. produces horizontal nystagmus to the right.

Douching the right ear with the head 120 degrees forward and the water at 68 F. produces horizontal nystagmus to the right.

Douching the left ear with the head 120 degrees forward and the water at 68 F. produces horizontal nystagmus to the left.

Douching the right ear with the head upright and the water at 112 F. produces rotary nystagmus to the right.

Douching the left ear with the head upright and the water at 112 F. produces rotary nystagmus to the left.

Douching the right ear with the head 60 degrees backward and the water at 112 F. produces horizontal nystagmus to the right.

Douching the left ear with the head 60 degrees backward and the water at 112 F. produces horizontal nystagmus to the left.

Douching the right ear with the head 120 degrees forward and the water at 112 F. produces horizontal nystagmus to the left.

Douching the left ear with the head 120 degrees forward and the water at 112 F. produces horizontal nystagmus to the right.

VERTIGO

Douching the right ear with the head upright and the water at 68 F. produces a sensation of falling to the left in the frontal plane.

Douching the left ear with the head upright and the water at 68 F. produces a sensation of falling to the right in the frontal plane.

Douching the right ear with the head 120 degrees forward and the water at 68 F. produces a sensation of falling to the right.

Douching the left ear with the head 120 degrees forward and the water at 68 F. produces a sensation of falling to the left.

Douching the right ear with the head back 60 degrees and the water at 68 F. produces a sensation of falling to the left.

Douching the left ear with the head back 60 degrees and the water 68 F. produces a sensation of falling to the right.

Douching the right ear with the head upright and the water at 112 F. produces a sensation of falling to the right in the frontal plane.

Douching the left ear with the head upright and the water at 112 F. produces a sensation of falling to the left in the frontal plane.

Douching the right ear with the head 120 degrees forward and the water 112 F. produces a sensation of falling to the left.

Douching the left ear with the head 120 degrees forward and the water at 112 F. produces a sensation of falling to the right.

Douching the right ear with the head back 60 degrees and the water at 112 F. produces a sensation of falling to the right.

Douching the left ear with the head back 60 degrees and the water at 112 F. produces a sensation of falling to the left.

PAST POINTING

Douching the right ear with the head upright and the water at 68 F. produces past pointing to the right.

Douching the left ear with the head upright and the water at 68 F. produces past pointing to the left.

Douching the right ear with the head back 60 degrees and the water at 68 F. produces past pointing to the right.

Douching the left ear with the head back 60 degrees and the water at 68 F. produces past pointing to the left.

Douching the right ear with the head forward 120 degrees and the water at 68 F. produces past pointing to the left.

Douching the left ear with the head forward 120 degrees and the water at 68 F. produces past pointing to the right.

Douching the right ear with the head upright and the water at 112 F. produces past pointing to the left.

Douching the left ear with the head upright and the water at 112 F. produces past pointing to the right.

Douching the right ear with the head back 60 degrees and the water at 112 F. produces past pointing to the left.

Douching the left ear with the head back 60 degrees and the water at 112 F. produces past pointing to the right.

Douching the right ear with the head forward 120 degrees and the water at 112 F. produces past pointing to the right.

Douching the left ear with the head forward 120 degrees and the water at 112 F. produces past pointing to the left.

FALLING

Douching the right ear with the head upright and the water at 68 F. produces falling to the right.

Douching the left ear with the head upright and the water at 68 F. produces falling to the left.

Douching the right ear with the head back 60 degrees and the water at 68 F. produces falling to the right.

Douching the left ear with the head back 60 degrees and the water at 68 F. produces falling to the left.

Douching the right ear with the head upright and the water at 112 F. produces falling to the left.

Douching the left ear with the head upright and the water at 112 F. produces falling to the right.

Douching the right ear with the head back 60 degrees and the water at 112 F. produces falling to the left.

Douching the left ear with the head back 60 degrees and the water at 112 F. produces falling to the right.

Galvanic Test: The test consists in applying a galvanic electrode to the mastoid with the indifferent electrode either on the other mastoid or in the patient's hand. A current of 3 to 5 milliamperes normally provokes responses.

The current may affect the end organ, the vestibular nerve or even the central nuclei, or it may affect all three, though about this there is some controversy.[33] It can affect the nerve, since responses to the exciting (cathode) stimulus may be obtained after a labyrinth has been destroyed and before the nerve has degenerated. It is in such a case that the galvanic test finds its greatest use, but as a matter of routine it has little application, because, as is generally agreed, this form of stimulation involves the entire labyrinth or at least is not specific.

Suffice it to say that the anodal stimulus is a depressing one and the responses are comparable to those obtained after removal of that labyrinth. The cathodal stimulus is an exciting one, and the responses are

33. Camis, M.: The Physiology of the Vestibular Apparatus, translated and annotated by R. S. Creed, London, Oxford University Press, 1930.

comparable to those obtained after removal of the opposite labyrinth. There is combined horizontal and rotary nystagmus, which does not change with change in the position of the head.

Fistula Test: If a fistula into the labyrinth exists and if suitable means are arranged [34] for applying compression and suction, responses may be obtained.

The effect of compression is nystagmus toward the affected side. Suction causes nystagmus to the opposite side. Compression and suction may be regarded as exciting and depressing stimuli, therefore, and they probably influence the labyrinth as a whole.

II. GENERAL COMMENT

In an approach to the problem of the vestibule, one fact, at least, remains undisputed. The labyrinth is an end organ which subserves an equilibrial function, the adequate stimuli of which are accelerated motion, the force of gravity and centrifugal forces. Whatever may be the interplay and coordination between the system of canals and the otolithic organ, peripheral, central or both, the adequate stimulus, at least for the canal system, is accelerated motion, while that for the otolithic organ is centrifugal force or the force of gravity. The canals are responsible for adjustment of tone to quick movements and to rotational movements. Stimulation of the canals induces nystagmus, past pointing, a subjective sense of rotation and falling. The otolithic organ governs the labyrinthine righting reflexes, the compensatory positions of the eye and the postural tone.

Only the vertebrates possess a canal system, the invertebrates being endowed only with an otolithic apparatus.

It would appear that the canals have evolved from the primitive otolithic apparatus for the purpose of providing an end organ more sensitive to sudden small movements in any plane of space. Given an end organ stimulable by a movement in any direction, the influence it will bring to bear on "muscle tone" through central integration is complex. Nevertheless, the end result must always be a purposive one.

Although they might seem unnecessary to mention, it is nevertheless important to keep two facts constantly in mind when studying vestibular responses:

1. The function of the vestibular apparatus during active or volitional movements is entirely different in end result from that demonstrated during the nonvolitional or passive experiment. In the voluntary act of looking to the right, not only is the head turned to the right,

34. Ruttin, E.: A Clinical Study of the Serous and Purulent Diseases of the Labyrinth, translated by H. Newhart, New York, Rebman Company, 1914.

but the eyes also make a wide and easy excursion to the right. The expected labyrinthine influence on the tone of the ocular muscles, demonstrated by passive experiment, must be entirely inhibited during such an event. Volition must always play a dominant role, while all static and tonic reflexes play a subservient role.

2. Static and tonic reflexes of any kind, including those of the labyrinth, are never imposed on the intact organism unconditionally, but are ever present to aid in the coordination of an act to the desired end. Labyrinthine reflexes are most prominent in the intact animal when being integrated with afferent stimuli from the proprioceptors toward a common end. Indeed, in the passive experiment, modifications of tone may not even exhibit themselves unless there is concomitant proprioceptive activity.[35] This was excellently illustrated by McNally when he demonstrated that tilting a frog whose belly was attached to a cork platform provoked no changes in the tone of its extremities. Yet as soon as its feet touched the table and gained kinesthetic contact with the inclined plane the necessary stabilizing changes in tone were immediately brought to bear.

Ewald [8] demonstrated that when the neck of a dog is turned, compensatory positions of the eyes are maintained as long as the animal tries to restore the position of the head. As soon as he gives up and relaxes the positions of the eyes are restored to normal.

Vestibular function has been painstakingly studied by many on the side of the end organ. The labyrinth has been stimulated and taken apart piece by piece and the facts recorded. With all such facts as a background, it is worth while to view the vestibule in a broader sense from the standpoint of purposiveness. If the desire is to fix the vision on some point, it will not be disturbed by bobbing the head about in any or all directions. The semicircular canals aid and in fact make possible the fixation. Yet, if the desire is to turn the head and eyes quickly to one side, the canals do not interfere.

Some have attempted to correlate the various canals more or less directly with specific extraocular muscles and thus account for the various forms of nystagmus. Nothing could be more divergent from the plan of the central nervous system. As Camis [33] pointed out, if it were true that each canal is functionally connected with a particular muscle, ocular nystagmus would necessarily vary its direction in the different positions other than it does, as the changes in position cause disturbance in the spatial correlation between canals and muscles.

In conformity with the view that each semicircular canal is in functional connection with all the muscles of the eye are the observations of

35. One exception to this rule, however, is the production of the slow phase only of nystagmus after a caloric stimulus during narcosis.

de Kleijn and Versteegh [17] on a rabbit which possessed only two canals and yet showed normal nystagmus in all directions.

I have observed that patients with palsy of the extraocular muscles present symmetric nystagmus after the rotation or the caloric test. Thus a patient with complete paralysis of the right sixth nerve, after calorization of either ear, presented second degree nystagmus in both eyes. Although the nystagmus in the right eye was much less intense than that in the left, it was nevertheless always of the same character.

Though normal nystagmus may be evoked from the decerebrate and decerebellate animal, it would be erroneous to assume that the cerebrum and cerebellum play no role in vestibular-ocular function in the intact animal.

When vestibular function has been viewed somewhat apart, it must then be dovetailed into the central nervous system as a whole.

The recently developed concepts of tone and posture marshaled by Magnus [9] and the Utrecht school and the monumental works of Sherrington [36] have aided greatly in an understanding of the integration of reflexes. The subject is profoundly important, but only those phases will be discussed that have a more direct relation to labyrinthine reflexes. Any movement, such as flexion of the arm, is, for every infinitesimal increment of change, subject to nervous influences that are occasioned by at least the posture of other parts of the body, the position of the head with respect to the body, the position of the head with respect to gravity and the vision, any or all of which may be in a state of change during the movement. The full appreciation of the mere fact that coordinated movement is the result of the perfect integration of so many complex reflex systems taxes the mind sufficiently, to say nothing of the complete darkness that obscures the ultimate mechanism by which such integration is accomplished.

I find it useful to regard an animal as having two positions, for which I use the terms "anatomic" which refers only to the spatial configuration during any instant, and "physiologic," which refers to tonal relations at the instant. The latter embodies the idea of movement either active or inhibited, and the direction or deviation of movement will always be toward the physiologic position. Such a concept finds easy application in studies of the labyrinth. The labyrinth, whatever may be the physical mechanism of stimulation, exhibits inertia. Hence, by severe stimulation beyond physiologic limits, a state is induced in which the physiologic position, so far as the role of the labyrinth is concerned, is markedly disassociated from the anatomic. Thus, in the

36. Sherrington, C. S.: (*a*) The Integrative Action of the Nervous System. New York, Charles Scribner's Sons, 1906; (*b*) Postural Activity of Muscle and Nerve, Brain **38**:191, 1915.

turning tests, if the subject is turned to the right, the physiologic position is disassociated to the left, and as long as this disassociation exists all voluntary and involuntary movement will deviate toward it. The eyes will deviate to the left (slow phase of nystagmus), and past pointing and falling will be to the left. Obviously the subjective sensation will be rotation to the right, and when rotation is suddenly stopped all phenomena will shift to the opposite direction. This hypothetic physiologic position, which is determined in part by labyrinthine impulses, always tends to restore to a zero position, but when disassociated from the anatomic it creates a state of tonal imbalance having definite characteristics, which is an exaggerated expression of the function of the labyrinth.

It has been customary in the Bárány tests to deduce what the normal reactions should be by reference to the canals in the plane of rotation, the reactions bearing a definite relation to the direction of the supposed flow of the endolymph. It seems far simpler merely to remember that the physiologic position is disassociated opposite to the direction of turning and reversed when turning is stopped. Other facts to be remembered are: 1. The plane of disassociation is always parallel to the floor after rotation but then remains fixed to the head in whatever position the latter may be placed. After rotation, when the head is moved in different positions, all responses induced by rotation will continue to bear a fixed relation to that plane of the head which was parallel to the floor during rotation.

As corollaries to this law, nystagmus, as Bárány pointed out, is always parallel to the floor during and after rotation, and its character, then, is unchanged regardless of the position of the head. The direction of the nystagmus and the plane of reference retain a fixed relation because both are fixed to the head.

Past pointing likewise becomes fixed to the plane of reference after rotation, which led Bárány to remark that it seemed as though past pointing became glued to the head. The modifications of past pointing when the position of the head is changed after rotation can be induced by stimuli from only two sources, the otolithic organ and the neck. If past pointing is induced to the left after turning to the left with the head upright, then turning the head to the left enhances the past pointing. Such movements of the head do not change the relation of the otolithic organs with respect to gravity but nevertheless influence the past pointing. These modifications of past pointing have their source undoubtedly in the reflexes of the neck. That past pointing is such a reflex—and not due, as Jones and Fisher [29] proposed, to conscious effort—is well substantiated by these facts alone. If a subject is past pointing to the right with eyes closed, as a conscious effort to compensate for his subjective sensation of rotation to the left, then it would be

incredible that rotation of the head on the body would modify the pointing—particularly to correct it entirely.

In defense of the role of consciousness in past pointing, Jones and Fisher [37] presented the following reasoning:

> Turning to the right with the head back 60 degrees produces past pointing to the right; turning to the right with the head forward 120 degrees also produces past-pointing to the right. If past-pointing were due to a cerebellar pull, this result would obviously be impossible; turning with the head backward and turning with the head forward produce diametrically opposite stimuli. If past pointing were a cerebellar reflex, the individual would in one instance point to the right and in the other instance point to the left. The past-pointing which occurs to the right in both instances, is because the cerebral centers receive the same impression of vertigo—that the body is turning to the left—no matter in what position the head may be placed.

In studying physiologic reactions, it is imperative that one always keep in mind the purposiveness of the response. The objective of any normal movement or reflex is that it serve a proper and useful purpose, and it will always be in the right direction. I should rather feel, with due deference, that this is an example of the pitfall that may confront one when he focuses attention specifically on one component of a complex machine—in this case, the vestibular end organ. Other influences are brought to bear on the raw stimulus from the labyrinth before the final efferent path is reached—particularly the reflexes of the neck and probably that of the otolithic organ itself. Though the stimulus of a vertical canal in one direction when rotation takes place with the head back causes past pointing in a certain direction, it does not follow by any means that therefore the response should be reversed because the stimulus is in the opposite direction when the head is forward. The business of an end organ is only to furnish a stimulus under certain conditions. The ultimate outcome of the stimulus will be integrated and adapted to a purpose.

Bárány (and Wittmaack) in 1911 ascribed to the otoliths the alterations which are observed in the pointing sign when the position of the head is changed.

The character of these reflexes makes possible the independent function of the head and body. For example, the hands may be caused to retain a stabilized position in front of the body in the same sense that the visual field may be stabilized against passive movements imposed in diverse directions. If it is desired to fix the visual field and the head is passively turned to the right, increased tone is reflexly induced in the proper ocular muscles so as to cause a compensatory deviation of the eyes to the left. In such a case the tendency would be to deviate the hands to the left were it not for the negating influence of

37. Jones and Fisher,[29] p. 188.

the reflexes of the neck. The latter reduce to zero any tendency to deviation. If, however, the neck and body are moved concomitantly, then the uninhibited tendency to deviate the hands to the left will be necessary to maintain their stabilization.

The whole plan of labyrinthine equilibration is designed to subserve such functions, namely, to aid in the stabilization of the body or any of its members against passive upsetting forces, particularly when it is the voluntary effort to stabilize, and other reflex systems, such as the visual and the proprioceptive, are incorporated in the integrating scheme toward that common end.

The considerations which have been given so far involve the function of the semicircular canals. Any movement which can be performed, with all its labyrinthine adjuncts, in the upright posture can be performed with practically equal coordination in any other position. Such changes in position introduce gravity as another variable, which necessarily alters the tonal relations in any antagonist-protagonist-synergist complex for the performance of equivalent movements. The otolithic organ, by throwing its influence into the integrating scheme, compensates for this variable.

Another pertinent fact merits general consideration. Although some of the static and dynamic reflexes do not manifest themselves objectively in the intact animal, they become conspicuous when forced to operate through their low reflex levels, disconnected from voluntary or cortical influences. Examples are the Magnus-de Kleijn reflexes of decerebrate rigidity.

It is rather interesting to consider the factors concerned in maintaining balance on a moving platform. Three situations involve the capacity for maintaining balance: (1) an attempt to balance an object on one's head or hand; (2) maintenance of one's own balance against diverse forces on an immovable platform, and (3) an attempt to maintain balance on a moving platform.

The first situation is rather simple, but those who have watched an acrobat balance a ladder on his shoulders and one perform on a tight wire will have noticed a striking contrast in the manner in which the two feats are performed. In the first case it is striking how stationary the ladder will appear to remain, while the acrobat's entire body makes corrective movements in all directions. Even more striking is the graceful manner in which a seal balances a ball on its nose. The head and neck of the seal move about, but the ball appears to remain fixed in space. The man on the tight wire, except when purposely leaning backward or attempting to recover a decided imbalance, maintains his head perfectly stationary, while the extremities and trunk engage in movement.

In the case of balancing an object, only two factors can possibly have direct importance—one, the sense of pressure, and the other, vision. When the center of gravity of the object deviates from dead center a diminished pressure is perceived and immediately corrected, the necessary direction of the correction being apprehended by vision. The objective of the acrobat is to keep getting under the center of gravity of his object. The vestibular apparatus plays no role here, except indirectly in maintaining the acrobat's equilibrium.

The second situation, which involves the actual function of the vestibular labyrinth, has already been discussed, but it is important to recall that in this case balance is affected through reflex resistance to displacement. Thus, if one is pushed to the right, a fall is prevented by an equal and opposite shift of intrinsic forces to the left.

It is the third situation that becomes intriguing and betrays the fact that the labyrinth was not designed to function on an unstable platform. In fact, the ability to maintain equilibrium on an unstable platform is proportional to the capacity for keeping the vestibules (semicircular canals) quiet and preventing them from entering into the reflex scheme. It is far easier to upset a person by shifting a rug under his feet than by attempting to push him over. The vestibular stimulus occasioned by shifting the rug evokes reaction in the wrong direction. The reflex resistance to a shift in his position can be made only with reference to the platform with which he is in kinesthetic contact, but this very resistance enhances the upsetting force.

It may be observed that a fun seeker at a fair who enters on to-and-fro-shifting platforms will maintain his equilibrium only so long as he can keep his head still and keep stimulation of the semicircular canals at a minimum. The tight rope walker prevents any shift of the head and forces all the balancing on the proprioceptive mechanism. The otolithic organ is important in correcting displacements of the center of gravity and in maintaining the upright posture, but sudden displacements must evoke in the semicircular canal responses at cross purposes; hence one learns to prevent movement of the head as much as possible.

A person without labyrinths and one on a shifting platform have something in common. Each finds his difficulties at a minimum if he can keep his head still. The person without labyrinths is disabled because movement of the head is unaccompanied by labyrinthine stimuli on which he formerly depended. The other is disabled because the passive movements of the head evoke reflexes that enhance the disturbance in equilibrium. Semicircular canals designed to function on a fixed platform cannot be expected to function properly on a shifting platform, and the reverse also would be true.

The question is often asked why the whirling dervish of the ballet or the ice skater does not experience extreme vertigo after rotation.

If one of these performers is carefully watched it will be observed that the head does not rotate concomitantly with the body but in a series of rapid complete turns to and from some fixed visual point, each followed by momentary fixation. (Helmholtz [38] made this observation but attributed its significance to ocular movements.) The sudden cessation of rotation of the head reverses or negates the labyrinthine effects of one complete turn. The entire rotary procedure therefore consists of interrupted rotation of the head, with labyrinthine stimuli annulled after each complete turn. The labyrinthine stimulus is not allowed to accumulate. The dancer educates himself to perform the rotation in this manner. With the novice or the subject in the Bárány chair, however, the uninterrupted rotation of the head allows the labyrinthine stimulus to accumulate.

Under "Psychic Adjunct of Labyrinthine Stimuli" I discuss the effects of violently and alternately rotating the head from side to side. The shifting visual field in this experiment indicates overstimulation of the semicircular canals but in alternate directions. Hence the end result is not vertigo.

III. SPECIAL PROBLEMS

PHYSICAL NATURE OF THE INNER EAR

Manner in Which the Receptors of the Canals are Stimulated.—As a result of the pioneering work and speculations on the manner in which the receptors in the cristae of the canals are stimulated, it was thought that rotation of the canals provoked currents in the endolymph. Although the possibility and probability of an actual flow of endolymph in the canals have remained in dispute, much evidence has been forthcoming to indicate that no flow occurs.

Although Mach [39] and Breuer [2] were early proponents of the theory of currents in the endolymph, Mach retracted after failing to produce comparable currents in glass models of the canals. Breuer likewise became skeptical and recognized that the hair cells are covered with a gelatinous material (cupula) and do not project, therefore, into the endolymph.

Maier and Lion [40] also studied the movement of fluids in capillary glass tubes and in the canals of the pigeon and the codfish and concluded

38. Helmholtz, H.: Helmholtz's Physiological Optics, translated from the third German edition, edited by P. C. Southall, Ithaca, N. Y., The Optical Society of America, 1925, vol. 3, p. 249.

39. Mach, E.: Physicalische Versuche über den Gleichgewichtssinn des Menschen, Sitzungsb. d. k. Akad. d. Wissensch. Math.-naturw. Cl. (Abt. 3) **68**: 124, 1873.

40. Maier, M., and Lion, H.: Experimenteller Nachweis der Endolymphbewegung im Bogengangsapparat des Ohrlabyrinthes bei adäquater und kalorischer Reizung: Physiologische Erklärung der Auslösung des Nystagmus durch Endolymphbewegung, Arch. f. d. ges. Physiol. **187**:47, 1921.

that the endolymph tends to remain at rest when the canals are moved, thus bending the cupula in the direction opposite to that of rotation. They point out the fact that stimulation for a fraction of a second can cause postrotatory nystagmus lasting twenty seconds. They postulate that some such chemical change must take place in the hair cells as is thought to occur in the visual receptors, producing an after-image.

Ewald [8] and Loeb [41] showed that section, ligation or plugging of the canals does not prevent normal responses.

Gray [42] called attention to the fact that the diameter of a canal in the squirrel is about one-fifth that in man, being about 0.75 mm. It is incredible that currents could be induced by turning such a miniature structure.

Ivy [43] endorsed Maxwell's theory that the receptors are stimulated by changes in pressure and tension and not by currents and that the cristae of the ampullae are stimulated by changes in the tension of the utricular membrane, which are in turn caused by the effects of inertia in the larger body of fluid in the utricle.

There are other experimental facts with which it is difficult to correlate existing views. For example, Maxwell [44] displaced the right horizontal canal of a dogfish to a vertical position without injuring the ampulla. When the dogfish was rotated on its dorsoventral axis to right or left, the compensatory eye movements were normal. Rotation of the animal around its longitudinal axis (in the new plane of the canal) did not produce a deviation of the eyes to the left as might be supposed. Maxwell postulated, therefore, that the sensory structures in the ampulla are stimulated in some other way than by currents in the endolymph produced by rotation.

Again, de Kleijn and Versteegh [17] reported that they had produced normal nystagmus in all directions in a rabbit which was subsequently proved to have only a horizontal and an anterior vertical canal on one side, with complete destruction of the labyrinth on the opposite side.

The semicircular canals have evolved from a more primitive otolithic organ, which itself is sensitive to movements, as well as to changes in position with relation to gravity. It would appear that an attempt has been made to evolve an organ that is not only more sensitive to

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41. Loeb, J.: Ueber Geotropismus bei Thieren, Arch. f. d. ges. Physiol. **49**: 175, 1891.

42. Gray, A. A.: The Labyrinth of Animals, Including Mammals, Birds, Reptiles, and Amphibians, London, J. & A. Churchill, 1907, vol. 1; 1908, vol. 2.

43. Ivy, A. C.: The Physiology of Vestibular Nystagmus, Arch. Otolaryng. **9**:123 Jan.) 1929.

44. Maxwell, S. S.: Labyrinth and Equilibrium: II. The Mechanism of the Dynamic Functions of the Labyrinth, J. Gen. Physiol. **2**:349, 1920.

movements but designed to reduce responses to greater specificity in any given plane. The canal system is stimulable in any plane, but being disposed in three separate planes it will be maximally stimulated in the plane in which it is disturbed—more so than would a spherical body of fluid.

It requires the greatest stretch of the imagination to assume that a current could be made to flow by mild rotation of a canal and that such a current would remain limited to the canal in which it is started. On the other hand, any medium, such as fluid, resting in a container will, if not rigidly fixed to the latter, change its pressure relations to the container in the event of acceleration. Such push and pull effects on the cupulae should be sufficient for stimulation of the cristae and would be more in keeping with the specificity in each plane for which the canals are designed.

Extralabyrinthine Reflexes Concerned with Equilibrium, Stabilization of the Visual Fields and Posture.—This heading incorporates the whole subject of righting reflexes and postural tone,[45] the investigations of which will not be recounted here.

De Kleijn (1918) found that by bending the neck or body of a delabyrinthized animal (dog or rabbit) or the tail of a dogfish he could produce compensatory movements of the eyes. The phenomenon was obliterated in rabbits when the dorsal roots of the first and second cervical nerves were sectioned.

Frenzel,[46] on the basis of his own observations and the literature, concluded that the reflexes of the neck in man extend to the eyes and appear only under special conditions. They act to produce compensatory positions of the eyes.

The various tropisms, such as heliotropism of insects, are striking examples of reflexes that are comparable to the righting reflexes in animals.

I should like to call attention to another type of reflex governing posture, which is probably conditioned on a cortical level. The reflex might be termed the "prepared posture reflex" for it is a reflex which prepares the person to meet an expected change in position or acceleration. It is obvious how such a reflex well timed could govern a more perfect balance. A person who has stood on the platform of a street car will be quite aware that when the car is expected to move forward one assumes a leaning posture timed to counteract the sudden acceleration. When the motorman closes the door and rings the gong but

45. Magnus.[9] Sherrington.[36b]

46. Frenzel, H.: Rucknystagmus als Halsreflex und Schlagfeldverlagerung des labyrinthären Drehnystagmus durch Halsreflexe, Ztschr. f. Hals-, Nasen- u. Ohrenh. **21**:177, 1928.

fails to start the car, one all but falls forward if not holding to some object. One learns subconsciously to time the conditioned reflex with events leading up to the acceleration.

Psychic Adjunct of Labyrinthine Stimuli.—Although an anatomic demonstration of vestibular pathways to the cerebrum is wanting, there is, nevertheless, physiologic evidence of the conscious representation of vestibular function.

Cyon [47] championed the hypothesis that the vestibule is an organ of the sense of space—a sixth sense. As Camis suggested, however, it is largely a matter of which way one wishes to view the problem. Stimulation of the vestibule calls forth many responses, which in the aggregate provide conscious information concerning the direction of movements and spatial relations. In this respect the vestibule is certainly an organ of the sense of space. The vestibule would be totally unnecessary to subserve the sense of space in the philosophic sense. The relative movement of any object at once establishes a concept of space, just as any change, chemical or physical, at once establishes a concept of time.

The fact that a sense of rotation and a sense of falling may exist in the complete absence of rotation or falling establishes a conscious representation of vestibular impulses.

The 2 patients previously discussed, for whom Dr. Dandy had sectioned the vestibular nerves bilaterally, presented reactions that betrayed a psychic adjunct of the vestibule. If when they were standing with eyes closed their heads were turned they felt that their feet rotated in the opposite direction. They were not conscious of the turning of the head. The proprioceptors in the neck provided the information that head and body changed relation, but this is only part of the necessary information. Also, if these patients suddenly turned their heads to look to the side or behind them, they became imbalanced without the subjective sensation of dizziness.

Although I have no objective proof of the following hypothesis, I should like to present a line of reasoning which has a bearing on the possible relation of the vestibule to the psychic interpretation of a stationary visual field.[48] The reasoning is founded on a proposition, or what might even be stated as a law. Whenever relation to the external world is changed, if the interpretation of the change is to be in keeping with the facts, it is necessary that information be received

47. Cyon, V., cited by Camis.[33]

48. After the preparation of this paper, I had an opportunity to study this phenomenon in my patient with torticollis, after section of one vestibular nerve. I was able to verify the hypothesis (Hyndman, O. R.: Torticollis Spastica: Suggested Etiologic Relation to the Vestibular Apparatus: Report of a Case. Arch. Otolaryng., to be published).

on a conscious level from receptors that are capable of being stimulated by the change in question. Otherwise an animal would have no means of differentiating between its own movements or changes in position and pseudoequivalent changes in its environment. There are numerous examples in common experience that bear this out—for example, the inability to tell whether one's own train or the train on an adjacent track is moving when the visual field is limited to both. When the eyes are moved about voluntarily, the perception is as it should be, namely, that the eyes have moved and the visual field has remained stationary. If the eyeball, however, is moved extrinsically, it appears that the visual field moves and not the eye. This is physiologic evidence of an afferent proprioceptive mechanism in the extraocular muscles. There is, of course, the possibility that the subjective impression received during voluntary movement of the eyes is integrated with the voluntary motor act entirely on a cortical level.[49] I have seen evidence of this possibility in tabetic patients with loss of proprioceptive function and in patients whose extremities had been deafferented, but such a function is inadequate by far to account for the perfection of the phenomenon in question.

Tozer and Sherrington[50] demonstrated the presence of sensory endings in the tendons of the ocular muscles.

Sense of the position of the eyes, as it were, has been referred to sense of the position of the visual field.

Such is the case when the eyes are moved about while the head is held stationary. Now, if the vision is fixed on some object and the head rotated from side to side, a new factor, the labyrinthine influence on the tone of the ocular muscles, is introduced. The tonal relations are, however, necessarily the same as if the eyes alone were moved, and consequently the subjective impressions are the same. If now the head is more vigorously rotated alternately from side to side, the visual field will appear to shift, always in the direction opposite to that of the rotation of the head. I feel that this phenomenon is an alternating example of that which occurs in the Bárány chair and is a manifestation of overstimulation of the semicircular canals.

If the head is rotated from side to side while the eyes are maintained passively at rest in the zero position, objects in the visual field remain

49. Helmholtz[38] (p. 243) evidently recognized this possibility. When he discussed fixation of the visual field and "muscular feeling," he mentioned three factors: (1) the intensity of the effort of will whereby one endeavors to bring the muscles into action, (2) the tension of the muscles and (3) the result of the effort.

50. Tozer, F. M., and Sherrington, C. S.: Receptors and Afferents of the Third, Fourth and Sixth Cranial Nerves, Proc. Roy. Soc., London, s.B **82**:450, 1910.

stationary. Their retinal images, however, shift across the retina with little or no concomitant activity of the extraocular muscles. Undoubtedly, impulses from another source must contribute to the proper psychic interpretation. Two possible sources are the labyrinth and the proprioceptive mechanisms of the neck.

One may perform an experiment that is even more striking. If one gazes straight forward, say at a picture on the wall, and then briskly abducts and adducts the head from shoulder to shoulder, one will probably find, as I did, that, though the object maintains its correct position, there is a decided tendency for it to seem to rotate counter to the direction in which the head is moving. In this experiment rotation of the eye on its anteroposterior axis causes the retinal image to move in a circular course on the retina. But here again the extraocular muscles contribute little, because the tendency of the eyeball to exhibit compensatory rotary movements in the frontal plane is almost negligible in man. The psychic compensation and adjustment probably must be attributed to some other source, and in this particular experiment, in which the other source appears to be entirely responsible, the adjustments are not so accurate as in those in which the ocular muscles can be used to some extent.

I believe, as a result of these experiments, that the psychic interpretation of movements and positions relative to the environment, particularly with reference to vision, is constructed of stimuli from several sources, viz., the vestibules and the proprioceptive mechanisms of the neck, the extraocular muscles and possibly the bodily musculature in general. It is believed that the labyrinth plays an extremely important role, and it would be expected that external objects would exhibit pseudomovements to those with bilateral absence of vestibular function, at least until the disparity is compensated by proprioceptive reflexes. I failed to question Dandy's patients on this point, but it is believed that a shifting visual field largely contributes to their imbalance on suddenly turning the head.

This may help to explain the bizarre postures that the monkeys of Northington and Barrera [23] assumed after bilateral labyrinthectomy. It seemed to make no difference whether the monkeys when they gazed at the examiner were right side up or upside down. It would seem that the ocular reflexes ought to aid in assuming the upright posture, but if every time the monkeys moved the visual fields shifted they might be expected to be satisfied with any position and loath to change.

Every one is familiar with the effect produced on the cinema screen by a moving picture taken on a rolling ship. The ship appears to remain stationary while the horizon appears to roll. The viewer sees the scene as the camera saw it. The camera, figuratively speaking, is not aware of its own change in position, and any change is interpreted

as an equivalent change in the environment. A person without vestibules should experience such a sensation.

The reverse of this phenomenon also sometimes occurs; i. e., by shifting the environment one may experience the illusion of movement. This is the well known experience in the "topsy turvy" houses of a fair. One sits in a swinging chair, and after the swing reaches a certain amplitude the room is rocked on a transverse axis in such a way that the amplitude of the swing appears to increase. Finally, by a complete overturn of the room, one is given a decided sensation of making a complete circle in the swing. This is an interesting experiment, demonstrating the limited accuracy of vestibulopsychic function.

Nystagmus.—Vestibular nystagmus has occasioned a great deal of thought. Many of the factors concerned remain sadly in need of satisfactory explanation.

The evidence at present is almost without exception in favor of the view that nystagmus is provoked by stimuli from the semicircular canals, the otolithic organ playing no role in its direct causation. The slow component is a result of increased tone homonomously and contralaterally imposed on the extraocular muscles. The quick component is a rapid return of the eyes to the position from which they were deviated. Arbitrarily nystagmus is referred to as in the direction of the quick phase. The slow phase of nystagmus (as induced by rotation) is an exaggerated exhibition of the normal influence of the labyrinth on ocular control. The labyrinth aids in making possible the stabilization or fixation of vision. If an attempt is made to fix the vision, movement of the head in any plane will be accompanied by a deviation of the eyes just sufficient to compensate for the displacement of the head.

Nystagmus produced by rotation is an exaggeration of the specific response that would occur within normal physiologic limits should the head be slightly deviated in the plane and direction of the rotation. There appears to be an exception to this rule in man. If the head were deviated in the coronal, or frontal, plane about an axis through the eyeball in an anteroposterior direction, then compensatory deviation of the eye would take the form of rotation about its anteroposterior axis. In exaggerated form, this is seen as rotary nystagmus and is produced by rotating the subject in the frontal plane (head forward 120 or backward 60 degrees). Although this type of nystagmus is easily produced, its normal counterpart consists of a maximum compensatory movement of only about 15 degrees with movements of the head. This compensatory movement in man is less, probably because of overlapping visual fields. It is prominent in the dogfish and the rabbit. Maxwell [5] showed that stimulation of the ampulla of the

anterior vertical canal in the dogfish caused the eyes to roll forward on their axes. Rotation in the plane of this canal produced the same results.

Such compensatory movements in the frontal plane can be measured in man by use of eyeball markers and cross lined glasses.[51] Combinations of this movement with compensatory movements in other planes are prominent in man, however, which makes the rotary deviation in pure form assume importance. For example, when the head is deviated laterally toward the shoulder fixation of vision requires that the eyeballs partially rotate in the orbital cavities, and the pupils will be seen to describe an arc with respect to the orbit. These combined forms of deviation may be produced in the form of nystagmus by rotation with the head placed in various degrees forward or backward.

Nystagmus is always in the plane of the canal that is stimulated (Flourens' law). The intensity of nystagmus is greatest in mammals. It cannot be demonstrated in the petromyzon or the turtle and is slight in the frog and the pigeon (Wilson and Pike [52]).

Nystagmus can be caused to operate normally through pathways that need course only in the brain stem. Bauer and Leidler (1911), Magnus (1916) and Ivy (1919) demonstrated normal nystagmus after removal of the cerebral hemispheres. Ewald (1892) found that in pigeons nystagmus was not affected by loss of the cerebral hemispheres.

Nystagmus occurs in a perfectly characteristic manner after removal of the cerebellum (Lange, 1891; Beyer and Lewandowsky, 1906; Wilson and Pike; [52] de Kleijn and Magnus, 1920). It is not abolished by injury to the corpora quadrigemina, although this does bring about disjunctive coordination of ocular movements (Wilson and Pike [52]).

The influence of vision and light on nystagmus has been discussed at length in the literature. Blind persons on rotation show vigorous nystagmus (Jones and Fisher [29]). Ohm [53] demonstrated that rotation in a dark room produces retarded "with" nystagmus of small amplitude and frequency and powerful "after" nystagmus of long duration. He concluded that the amplitude of nystagmus produced in the presence of light is considerably greater than that produced in darkness.

Woolsey,[54] comparing blind with normal pigeons, concluded that vision exerts a controlling influence over vestibular function. He found that amblyopia in pigeons diminished nystagmus during rotation.

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51. Camis,[33] p. 222.

52. Wilson, J. G., and Pike, F. N.: The Mechanism of Labyrinthine Nystagmus and Its Modifications by Lesions in the Cerebellum and Cerebrum, Arch. Int. Med. **15**:31 (Jan.) 1915.

53. Ohm, J.: Bessere Kurven des vestibulären Drehnystagmus, Ztschr. f. Hals-, Nasen- u. Ohrenh. **19**:467, 1928.

54. Woolsey, C. L.: The Influence of Vision on Nystagmus, Ann. Otol., Rhin. & Laryng. **33**:235, 1924; Influence of Vision on Nystagmus, Arch. Neurol. & Psychiat. **7**:526 (April) 1922.

It is noteworthy that Ewald,[3] while demonstrating compensatory ocular movements in labyrinthectomized dogs, observed a few nystagmic jerks when bending the neck of the dog. He observed nystagmus also in pigeons without labyrinths during rotation but no "after" nystagmus. No nystagmus occurred when a pigeon without labyrinths was placed in a cylinder of gray paper and the field of vision rotated with the bird. Normal pigeons exhibited practically no nystagmus while turning in the gray cylinder but had definite "after" nystagmus.

All such experiments demonstrate that while the labyrinth exerts a powerful influence on stabilization of the visual field, the reflexes of the eye and probably of the neck play some role. This is not surprising, because one would expect that all functions, such as vision and turning of the neck, which bear a close relationship to ocular movements would develop associated reflexes subserving fixation of vision.

Undric[55] found that after median bisection of the floor of the fourth ventricle he could not cause nystagmus either by calorization or by rotation. Paramedian section, however, provoked spontaneous nystagmus, which was arrested by calorization. Undritz[56] obtained spontaneous nystagmus by paramedian section and found that he could produce typical nystagmus after median section. Both authors agreed that the minimum rotation perceptible to the vestibular apparatus is an angular rotation of 3 degrees per second.

The feature of nystagmus which has been most disturbing is the quick component. There has been no satisfactory explanation of its mechanism.

That the quick phase does not depend on cortical function is well proved by the fact that characteristic nystagmus can be produced after removal of the cerebral hemispheres.

The quick phase may be eliminated by narcosis while the slow phase is still manifest. The slow phase can be produced alone in sleeping children by rotation, and it can be elicited alone for some time after birth.[52]

The presence of the quick phase in the blind eliminates the visual reflexes as a cause.

If one labyrinth is destroyed and some weeks later the other is destroyed, characteristic nystagmus is produced temporarily to the side opposite that last operated on, as if the first labyrinth were intact. This

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55. Undric, V.: Zur Frage über die nystagmatischen Bewegungen isolierter Augenmuskeln, Russk. oto-laring. **21**:5, 1928; abstracted, Zentralbl. f. Hals-, Nasen- u. Ohrenh. **12**:535, 1928.

56. Undritz, W.: Ueber den Einfluss verschiedener Versuchbedingungen auf den Drehnystagmus und über die Bedeutung der contralateralen Verbindungsfasern des N. vestibularis. Ztschr. f. Hals-, Nasen u. Ohrenh. **19**:444, 1928.

reaction is known as Bechterew's compensation and demonstrates that the quick phase does not have its source in the vestibular end organ itself.

In fact, there is practically general agreement that, just as the pathways involved in the production of the slow phase need be only in the brain stem, so the source of the quick phase also need be only in the brain stem.

Questions that naturally arose were: 1. Is the quick phase due to active muscular contraction or primarily to relaxation of the tonic muscles or to both? 2. Does the reflex involved depend on afferent impulses from proprioceptors in the extraocular muscles?

Bartels [57] dissected the rectus internus and externus muscles free, so that they could function independently, and by means of kymographic records demonstrated that during nystagmus when one muscle is relaxed the antagonist contracts. Whatever the mechanism, it exhibits reciprocal innervation. The experiment also indicated that the relaxation of the contracted muscle is definite and independent of its antagonist.

De Kleijn [58] prepared an animal by sectioning the brain anterior to the corpora quadrigemina and cutting the third and fourth nerves bilaterally and the sixth nerve on the right. Only the sixth nerve on the left remained intact. The left rectus externus muscle was connected with a kymograph and was shown to exhibit both slow and quick phases. He attempted to anesthetize only the afferent fibers with procaine hydrochloride, but this procedure stopped both phases simultaneously. The experiment is important enough to merit careful checking. It is the one experiment which was designed to prove or disprove the possibility of proprioceptive influence on nystagmus.

Lorente de Nó [59] showed that the rapid phase can be eliminated with preservation of the slow phase by lesions in the reticular substance. He [60] stated also that as a result of his experiments it appeared that the development of the rapid phase, as Bárány asserted in 1907, is dependent on intactness of the reticular substance in the pons and that

57. Bartels, M.: Ueber Regulierung der Augenstellung durch den Ohrapparat: III. Kurven des Spannungszustandes einzelner Augenmuskeln durch Ohrreflexe, Arch. f. Ophth. **78**:129, 1911.

58. de Kleijn, A.: Experiments on the Quick Phase of Vestibular Nystagmus in the Rabbit, Versl. d. wis- en natuurk. Afd. d. k. Akad. v. Wetensch. **29**:1230, 1921.

59. Lorente de Nó, R.: Ueber den Nervenmecanismus der vestibulären Augenbewegungen, Acta oto-laryng. **12**:243, 1928.

60. Lorente de Nó, R.: Untersuchungen über die Anatomie und die Physiologie des Ohrlabyrinthes und des Nervus octavus: Die Labyrinthreflexe auf die Augenmuskeln nach einseitiger Labyrinthexstirpation nebst einer kurzen Angabe über den Nervenmechanismus der vestibulären Augenbewegungen, Monatschr. f. Ohrenh. **61**:1066, 1152 and 1300, 1927.

the neurons from which the rapid phase is evoked must be connected with the vestibular nuclei.

Wilson and Pike [52] found, unlike other investigators, that complete decerebration of cats and dogs, including elimination of the corpora striata and the thalamus, wholly abolishes the quick component (after stimulation or removal of a labyrinth), although the slow deviation persists. They concluded that the quick phase arises from some mechanism above the corpora quadrigemina.

I should like to propose a possible explanation of the quick phase along the following lines: It must be remembered that nystagmus is produced by an excessive stimulus and that its mechanism is mediated in subcortical levels. A close analogy may be drawn to ankle clonus. If one is to obtain an extensor response (extensor being used in the physiologic sense) the extensor muscles of the ankle must be stretched. This stimulus is followed by involuntary contraction which, if allowed to occur, becomes the adequate stimulus for release. If the original stimulus, that is, the forceful dorsiflexion of the foot, is maintained, the response proceeds in the characteristic clonic rhythm. Ankle clonus does not appear in man after a complete transverse lesion of the cord.[61] Likewise, if a lesion of the brain stem occurs in the location which gives rise to decerebrate rigidity, the extremity is maintained with the extensor muscle in a state of continued rigidity, and here again clonus cannot be obtained.[62]

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61. Dr. Clarence Van Epps, of the department of Neurology, College of Medicine, State University of Iowa, recalls a case of complete transverse lesion of the cord, in which clonus of the ankle and patella was clearly demonstrated. The patient was a carpenter aged 32. Ten years previously he fell and sustained a fracture of the spine. A complete transverse lesion of the cord was suspected. When examined he presented spastic paraplegia (4 plus) with hyperactive deep reflexes and sustained clonus of the ankle and patella. There was an extensor response (Babinski) on both sides. Sensation was completely lost below the sixth thoracic segment. Laminectomy was done, and the cord for a length of about two segments was found to be shrunken and scarred. The patient died twelve hours after operation. Sections stained by the Weigert-Pal method revealed a total loss of myelin and axis-cylinders in the scarred segment. The cord above and below the lesion did not appear unusual except for degeneration of ascending tracts above and of descending tracts below.

This was an unusual case, but perhaps only because it is rare for a patient to live so long after a complete transverse section of the cord.

62. There is some question concerning the existence of clonus after decerebration. H. Viets (Relation of the Form of the Knee Jerk and Patellar Clonus to Muscle Tonus, Brain **43**:269, 1920), working with decerebrate cats, found that true clonus could be obtained if his crural-vasticrureus preparation was in a state of "neutral tension." If the tonus was varied from this state by stimulation of a

(Footnote continued on next page)

If the lesion is more cephalad, i. e., of the pyramidal system in man, clonus appears. It seems evident, therefore, that clonus is a rhythmic reflex composed of two elements: first, reflex contraction of the extensor muscles, the adequate stimulus of which is a stretch, and, second, reflex release of contraction, the adequate stimulus of which is contraction of the muscle. It may be easily observed how forceful clonus may be. All the powers of muscular contraction are brought to bear until shortening of the muscle is accomplished, and then suddenly all the contracting force is completely released. Deafferentation abolishes the phenomenon.

It is not too imaginative to see the same mechanism at work in nystagmus. The maintaining force here (comparable to the examiner's

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peripheral nerve, the clonus was abolished. He did not always find it. He also observed pseudoclonus in the spinal animal.

I have encountered a number of human beings who I have felt were affected with decerebrate rigidity or a condition closely related to it. One was an infant with an abscess of the right frontal lobe. The Magnus-de Kleijn reflexes of the neck could be excellently demonstrated.

Another was a child who had a spongioblastoma in the right corpus striatum, with a small extension to the suprasellar region that was mistaken for a primary suprasellar tumor. During the removal of the latter a clip was inadvertently placed on the left anterior cerebral artery. Although before the operation the child exhibited no signs referable to the striatum, after operation she presented extreme rigidity of the extensor muscles of the right upper and lower extremities, with the forearm markedly pronated and the ankle and toes markedly extended.

Another child, with a tumor limited to the superior colliculus and marked hydrocephalus, who was hypotonic before operation, after operation presented marked rigidity of the extensor muscles of all four extremities. The reflexes of the neck could not be demonstrated. The vermis had been divided entirely in the midline.

These patients never regained consciousness. Whether they would have fulfilled the postulates of F. M. R. Walsh (The Decerebrate Rigidity of Sherrington in Man: Its Recognition and Differentiation from Other Forms of Tonic Muscular Contraction, Arch. Neurol. & Psychiat. **10**:1 [July] 1923) or not is a question, but I feel that, so far as tone is concerned, the rigidity of the extensor muscles was of the decerebrate type.

Clonus could not be obtained in these cases. In fact, the extension of the ankle was difficult to overcome, although the ankle and knee would relax under stress.

I hope to present these and other cases in detail in another paper, to which they rightfully belong, but the key to the situation seems to have been in the absence of a mechanism for release—hence the maintained extension.

I feel that as one begins to take away the supranuclear structures of the brain decerebrate rigidity is approached from the beginning but not rendered maximum in lower animals until everything cephalic to the red nucleus, including the cerebellum, is removed (section entitled "Role of the Cerebellum in Decerebrate Rigidity"). But in man it is likely that less complete releases provoke states of rigidity of the extensor muscles that operate through the same mechanism as decerebrate rigidity.

hand in dorsiflexion of the foot) is the stimulus from the labyrinth which continues to impose an increased tone in the extraocular muscles involved. When the muscles have shortened sufficiently, the stimulus for release from contraction is given through the afferent connections from the proprioceptors, and the quick component follows. The labyrinthine stimulus for increased tone is still present, however, and the process assumes a rhythmic nature. The reciprocal function of the antagonists here could logically be due to a central reflex, but the release is the primary and necessary factor in initiating the quick phase.

This hypothesis involves the supposition of two reflex arcs, one subserving the contraction phase and one the release phase. It appears that the latter is more cephalad than the former and can be eliminated as an independent reflex.

In substantiation of the hypothesis, it might be recalled that voluntary deviation of the eyes and conjugate deviation associated with an epileptic seizure are not nystagmoid if the cerebellum is intact. To be clonic, the reflex must be occasioned through subcortical levels. The elimination of the quick phase by decerebration (Wilson and Pike [52]) strengthens the analogy to ankle clonus.

Also the observations of Lorente de Nó concerning elimination of the quick phase by lesions of the reticular substance are interesting in this light.

In spite of the general uniformity of opinion and of experimental results to the effect that removal of the cerebrum and cerebellum does not abolish characteristic nystagmus, there have been various reports of modifications of nystagmus due to lesions of the cerebrum and cerebellum.

Grahe [63] reported observations on a patient in whom the right cerebellar hemisphere was absent and concluded that removal of the cerebellum makes nystagmus less excitable than in the normal subject. In his patient nystagmus was more intense when rotation was to the right. Calorization of the left ear, after one minute, produced nystagmus to the right. Calorization of the right ear, after fifteen seconds, produced nystagmus to the left, lasting three minutes, accompanied by past pointing to the right, especially with the right hand, which was ataxic.

Démétriades and Spiegel [64] by removal of one labyrinth produced nystagmus to the sound side. After the stage of compensation was reached, cauterization of the cerebellar cortex on the side of the

63. Grahe, K.: Kann vom Kleinhirn Nystagmus ausgelöst werden? Ztschr. f. Laryng., Rhin. **17**:99, 1928.

64. Démétriades, T. D., and Spiegel, E. A.: Zur Frage d.. Bedeutung des Kleinhirns für die Entwicklung von Spontannystagmus, Ztschr. f. Hals-, Nasen- u. Ohrenh. **19**:250, 1927.

operation, without involvement of the cerebellar or the bulbar nucleus, produced nystagmus directed to the same side.

Again Wilson and Pike [52] stated that removal of a cerebral hemisphere, vermis or cerebellum may affect but does not abolish the nystagmus that follows destruction of a labyrinth. They showed that removal of a cerebral hemisphere on the side of the slow phase abolished all except a small rotary component of the quick phase. Removal of the hemisphere on the side of the quick phase had no effect.

Jones and Fisher [29] presented a number of cases of tumor of the cerebellopontile angle in which the ipsilateral labyrinth was dead and no response could be obtained from calorization of the vertical canals of the contralateral labyrinth. A good response was obtained from the horizontal canal. From these observations Jones postulated that the fibers from the horizontal and the vertical canal follow a different course to and in the brain stem. There is, however, no anatomic verification of this hypothesis.

Selling and Kistner [65] presented a case of tumor of the cerebellopontile angle on the right, with loss of all vestibular responses on the same side. Calorization of the vertical canals on the opposite side (head 30 degrees forward) for three minutes caused cessation of spontaneous nystagmus to the left. They quoted H. T. Lillie as stating that in only 1 of his 6 cases of tumor of the angle was the Jones syndrome exhibited.

Most observations of this nature have been made on clinical material, and views have been expressed on the value of atypical, perverted and reverse nystagmus and other vestibular responses in the diagnosis and localization of tumors or lesions of the cerebrum or cerebellum.[66]

It is usually precarious to draw rigorous physiologic conclusions from cases of tumor, especially if the growth is massive or causes any significant compression. Conclusions drawn from observations in such cases vary a great deal as recorded in the literature, even in apparently similar cases. I have applied the usual vestibular tests in cases of cerebellar or vermian tumor and at present I doubt that the tests offer any dependable aid in localization except, of course, in the differentiation of the peripheral and the central mechanism. The whole subject is important, however, and more properly belongs to another chapter.

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65. Selling, L., and Kistner, F. B.: The Labyrinthine Tests in Cerebellar Diagnosis, J. A. M. A. **81**:1194 (Oct. 6) 1923.

66. Blonder, E. J., and Davis, L.: The Galvanic Falling Reactions with Verified Intracranial Neoplasms, J. A. M. A. **107**:411 (Aug. 8) 1936. Coates, G. M.; Shuster, B. H., and Slotkin, H. B.: Vestibular (Bárány) Tests in the Diagnosis and Localization of Intracranial Lesions, J. A. M. A. **107**:412 (Aug. 8) 1936.

Labyrinthectomy.—Bilateral labyrinthectomy will not be discussed further, but the phenomena following unilateral labyrinthectomy are still problematic, and certain features merit further discussion.

In the section concerned with the responses following labyrinthectomy in various animals, it will be noted that certain responses are strikingly common to all the animals studied. Also certain reactions disappear early, while others remain permanently.

Camis [67] listed the effects of unilateral labyrinthectomy in the dog as follows:

A. *Irritative signs.*

Rolling movements around the longitudinal axis.
Circling movements.
Gyratory movements.
Ocular nystagmus.

B. *Deficiency phenomena, not modified by time.*

Torsion of the head.
Difficulty in jumping down from a height.

C. *Deficiency phenomena, compensated as time goes on.*

Contraction of the pupil on the operated side.
Exaggerated side to side movements of the head.
Tendency to fall to the operated side.
Inability to stand on the hind legs.
Stepping gait.

There has been some controversy concerning whether the temporary signs are irritative or deficiency signs. Although Camis recorded certain of them as irritative signs,[68] the consensus is that they are not irrita-

67. Camis,[33] p. 61.

68. The temporary active, or exalted, signs ensuing on elimination of a labyrinth are extremely interesting and important from a physiologic standpoint and must be accounted for. There are three possible explanations:

1. They are irritative in the sense that the cut end of the vestibular nerve is in a state of active irritation.

2. The opposite labyrinth assumes a state of overactivity.

3. The removal of a labyrinth constitutes of itself alone an absolute "minimal" or "negative stimulus."

At first the third concept might appear fantastic, but it is far from being too conjectural. If the labyrinth is an end organ capable of being excited or depressed—and that is what all the facts demonstrate—then, when supposedly at rest, it imposes a controlling influence on its primary nuclei. An adequate depressing stimulus normally acts reciprocally with an exciting stimulus from the opposite labyrinth. The "depressing stimulus" might at first be classed as

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tive. It would seem that the experiment of König (1897) and of Gaglio (1899) leaves little doubt that the signs are due to a deficiency of the end organ. These workers showed that after cocainization of the labyrinth, which constitutes paralysis without irritation, the signs were the same as when the labyrinth is ablated or the vestibular nerve sectioned.

Camis [69] based his belief in the probability of irritation on the fact that he had shown (1911) that oscillations of tonus in the gastrocnemius muscle of the frog are abolished when cocaine is introduced into the stump of the vestibular nerve after labyrinthectomy. The phenomenon is ipsilateral.

At present it would seem difficult to escape the belief that the signs ensuing when one labyrinth is removed are due to relative overactivity ot the remaining labyrinth. The nystagmus and other signs are in the proper direction from this point of view and in the wrong direction if caused by operative irritation. This belief is in keeping with the galvanic responses. For example, the anodal current to the ear causes the same responses as the removal of that labyrinth, and the anodal current is known to be a depressing stimulus. The cathodal current causes the signs to reverse their direction.

While it is not as rigorous evidence, it is nevertheless pertinent that the cold stimulus in the caloric test causes signs in the same direction as the anodal current or labyrinthectomy, while the hot stimulus causes signs in the reverse direction. The caloric reaction will be discussed further, but I feel that cold is a depressing stimulus.

All the facts seem to indicate, as brought out before, that the two vestibules function reciprocally, each being capable of excitation or depression. Depression of one labyrinth is equivalent to excitation of the other. But that depression of a labyrinth in the absence of the other can constitute a stimulus, in the negative sense, is demonstrated by Bechterew's compensation.

Labyrinthectomy, the galvanic and the caloric test do not constitute natural stimuli, but rotation does, and in the case of rotation the paired organ functions reciprocally, one being depressed while the other is excited, according to the direction of rotation. The labyrinth toward

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inhibition, but it cannot fit the present befogged concept of inhibition, because the facts demonstrate that the responses are due, not to original active impulses, but to an actual decrease in the number of incoming stimuli. Labyrinthectomy constitutes the maximal decrease. This "releases" the primary nuclei to impose their unbridled influence on muscular tone just as if the labyrinth were intact but in a sustained state of minimal stimulation. This is an example of a decrease in or absence of afferent stimuli seeming paradoxically to function as active stimuli. Within a few days, the exalted, or active, signs wear away, leaving responses which exhibit themselves only during an attempt to move and which are clearly referable to the failure of incoming corrective stimuli.

69. Camis,[83] p. 65.

which the head is turned is excited and provides the dominant stimulus. This occasioned Ewald to state that the excited labyrinth (endolymph flowing toward the ampulla) provides two thirds of the stimulus while the other provides one third.

In man, when one labyrinth is destroyed the other can soon be demonstrated to function bilaterally, but the intensity of the signs is unequal after rotation to the right and to the left. In time (about a year) compensation occurs and the intensity of the signs approaches equality.

It would seem that the mere fact that the signs can be reversed in direction by the anodal or the cathodal current or by stimulation with heat or cold is strong evidence that one labyrinth can be excited or depressed and in either case provide a well defined ultimate stimulus.

Although it was stated in the foregoing discussion that after removal of one labyrinth the other is in a state of relative overactivity (and this appears to be the consensus), I am inclined to believe that the temporary responses, i. e., nystagmus, vertigo, past pointing and falling, are due to a negative stimulus from the dead labyrinth. Although the intact labyrinth is still subject to its own exciting and depressing stimuli, strictly speaking it has nothing to do with the case. If it is treated with cold water or the anodal current or removed, the nystagmus is diminished or abolished. If it is treated with hot water or the cathodal current, the existing nystagmus is enhanced. All the responses that the intact labyrinth subserves are superimposed on the responses that are due to the recently destroyed labyrinth.

After unilateral labyrinthectomy the responses are of two kinds:

1. Those due to the absolute negative or depressed stimulus—nystagmus, vertigo, past pointing and spontaneous falling. These are probably entirely due to loss of the semicircular canals and disappear in a few days as adaptation quickly takes place.

2. Those due to the absence of corrective afferent stimuli—the incoordination during movement.

The vertigo or subjective sense of dizziness, as I demonstrated previously, is replaced by a sense of imbalance without dizziness. The sense of imbalance has the same cause as the motor incoordination and does not disappear until the intact labyrinth fully compensates, acquiring a bilateral function.

Torsion of the head requires separate consideration, for it would appear that this is the one permanent manifestation of loss of an otolithic organ. This phenomenon is noted immediately after operation; not only does the head assume a position with the dead labyrinth dependent, but the tonus in the extremities simulates that which would be assumed if the animal were subjected to centrifugal force in a direction from the dead to the intact labyrinth. The depression of

one otolithic organ creates such a situation and is partly compensated by depressing the stimuli from the intact otolith. This is accomplished by turning the head so that the intact otolithic organ is uppermost. Such a rotated position of the head constitutes a new normal posture, any deviation from which provides a sensation of imbalance with respect to gravity. The animal or human being is powerless to correct the immediate effects of loss of the canals but he can by so maneuvering the head minimize the effects of the loss of an otolithic organ. Therefore, a patient with a tumor of the cerebellopontile angle which has destroyed the vestibular nerve deviates the head in such a way as to bring the intact otolithic organ into a superior position.

I do not wish to imply that the change in position of the head is entirely voluntary but that it is a reflex forced position in the same sense as is the righting of an animal's head after it has been displaced from the normal upright posture.

Breuer [70] considered rotation of the head following extirpation of a labyrinth a response determined by the otolithic organ.

In summary, all the observations seem to indicate that each semicircular canal is in functional connection with both sides of the body and that the direction of responses depends on whether the crista is maximally or minimally stimulated, or, in another sense, on whether the stimulus is positive or negative. The destruction of the canals on one side constitutes an absolute minimal stimulus, which lasts several days to a week. It is irritative only in the negative sense. After this stimulus wears off and compensation occurs, all the responses to rotation or to calorization can be produced by the other canal system, which is thus proved to be bilaterally represented and responsive to maximal and minimal stimulation. It appears that all the short-lasting phenomena are referable to the canal system.

The otolithic organs also function bilaterally and reciprocally, but the loss of an otolith appears more to upset the gyroscopic balance of the paired organ. This imbalance is compensated to some extent by torsion of the head, which places the intact otolith in an optimum position to subserve a balanced tone on the two sides and is more or less a permanent posture.

Long-lasting incoordination of movements is a result of the absence of corrective afferent impulses in the event of movement. In time the intact labyrinth will become largely, if not entirely, adapted to a balanced bilateral function.

I feel that the oscillations of the head in some animals which have been described are due to the absence of corrective afferent stimuli from the canals. The oscillations of wide amplitude are expressive of loss of the canals and indicate that the canal system normally exerts a steady-

70. Breuer, cited by de Kleijn.[72]

ing influence on the head. When the animal is aroused from a relaxed position, the absence of this influence is manifested.

Double Role of the Inner Ear and the Interrelation of Its Functioning Parts.—As has been brought out before, the semicircular canals have probably evolved from the primitive otolithic organ and provide a mechanism of far greater sensitivity to minimum changes in position. Experimental results have seemed to point to a rather sharp division of labor between the canals and the otolithic organ. It is generally agreed that the canal system when stimulated by rotational movement or accelerated displacement induces nystagmus, past pointing, a subjective sense of rotation and falling. The otolithic organ governs the righting reflexes, compensatory ocular positions and postural tone, the adequate stimulus being gravity or centrifugal force.

In man the paired canal systems and otolithic organs seem to function in a reciprocal manner and are subject to maximal and minimal stimuli. The maximal stimulus of the canals causes homonomously an increase in tone of the contralateral musculature and is designed in this way to resist a rotary displacement. Each otolithic organ governs the musculature bilaterally, and the two organs function reciprocally, each being subject to maximal and minimal stimuli. It appears that if the ears are placed one above the other with respect to the ground the dependent otolithic organ is maximally stimulated and the other is minimally stimulated.[71] There is some controversy on the point, but the evidence seems in favor of this view. Maximal stimulation causes rotation of the head to the ipsilateral side and increased tone of the extensor muscles in the ipsilateral extremities, associated with abduction. Minimal stimulation causes the reverse. The posture is designed to counterbalance a centrifugal or gravitational force. For example, the safest and easiest posture for a person on the periphery of a turning table is one in which his coronal plane is in line with a radius of the table; he will lean toward the center, with the outermost lower extremity extended and abducted. Animals on all fours will extend and abduct both upper and lower outermost extremities.

Reciprocal function of the paired canal systems does not exist in certain of the lower animals. If one labyrinth is destroyed in the dogfish, normal responses will result when the animal is turned to the sound side but none when it is turned to the side of the operation. McNally demonstrated this phenomenon also in the frog. In man, however, as is well known, the canals on one side will cause responses when the subject is turned in either direction, and in time the intensity

71. "Maximal" and "minimal" are used relatively here. It will be recalled that maximum bilateral tone of the extensor muscles exists when the animal is supine and minimal when it is prone. Varied position in the frontal plane will exhibit differences in the tone of the extensor muscles on the two sides.

of the responses occasioned by turning in either direction will approach equality, the destroyed labyrinth being then fully compensated.

It will be remembered that in the presentation of the physiologic facts it was brought out that according to McNally the unilateral phenomena in the frog indicate that the cristae are stimulated by movement of endolymph in only one direction. I feel that the possibility of bilateral representation in man and unilateral in the frog admits of an alternative explanation.

It is clear that however sharp may be the division of labor between the canals and the otolithic organ, their respective functions must dovetail nicely to subserve the purpose of the vestibule.

De Kleijn [72] stated the belief that progressive reactions occur through a combination of reflexes arising both in the semicircular canals and in the otoliths.

The idea of "minimal" stimulation of the canals provides the only adequate explanation of certain strange phenomena. That is, if the end organs are regarded as being in a constant "basal" state of stimulation, even when the organism is at rest, then a physical change can be brought about in the canals with respect to the endolymph that results in a diminution of afferent stimuli or a "negative" stimulus. This negative stimulus, so far as responses are concerned, would be equivalent to a positive stimulus from the opposite side. Thus, several weeks after a labyrinth is destroyed, the temporary phenomena, such as nystagmus, will have disappeared, and compensation will have taken place. If the other labyrinth is now destroyed, nystagmus will appear to the side of the first operation as though that labyrinth were intact. This phenomenon is known as Bechterew's compensation and appears to be due to the negative, or absolute minimal, stimulus occasioned by destruction of the second labyrinth.

Independence of the Separate Canals: It has been shown that normal responses of the canals can be elicited in animals after the utricles have been eliminated.

That stimulation of a single canal will produce nystagmus in the plane of that canal is a proved fact (Flourens' law). Tait and McNally [6] eliminated individual canals in frogs and showed that the defective responses were in the plane of the destroyed canal or canals.

In the rotation tests it is undoubtedly the canal in the plane of rotation that is stimulated, at least maximally with respect to the other canals.

All the facts known would lead one to believe that the individual canals are fixed and inseparable from their correlated responses and that if any are absent their respective responses should be wanting.

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72. de Kleijn, A.: Experimental Physiology of the Labyrinth, J. Laryng. & Otol. **38**:646, 1923.

Nevertheless, there are situations which appear to contradict this belief. In Maxwell's dogfish a normal response was elicited in spite of the displacement of the horizontal canal to the vertical. Why there was no response when the animal was rotated in the vertical plane is difficult to explain. One may recall also de Kleijn's rabbit with only two canals on one side which nevertheless exhibited normal nystagmus in all planes.

I am not aware of a case on record in which there was known destruction of only one canal in a human being.

At any rate, one might look on the canal system as an end organ which is stimulable by movement in any plane. Given such an end organ, all its responses will be integrated centrally in order that it may most purposefully subserve its end. Hence, if one displaced the entire end organ, readaptation would be expected in the course of time. Also, if a canal were destroyed, whereas the maximal stimulus would be severely crippled, there would not necessarily be entire absence of stimuli when the subject was moved in that plane. Such stimuli as would be forthcoming would be readapted in time to subserve the purposeful end, and it is not inconceivable that, after due time for adaptation, the absence of a canal might be hardly detectable. Indeed, the vertical canals are placed eccentric to the planes in which the head primarily moves, namely, the sagittal, in nodding affirmatively, and the coronal, or frontal, in abduction and adduction. Also the canals are not precisely at right angles to each other, as is evident in some animals—for example, the pigeon.

Are the canals represented separately in their central connections?

There is no histologic evidence that the canals are separately represented. Each canal, however, has its own group of receptors in the crista, from which emerges a distinct bundle of axons.

The fact that when a canal is eliminated responses fail to occur in the plane of that canal (as McNally found in frogs) would not per se force the conclusion that the canals have independent central representation. If the end organ were defective in a given plane, then afferent stimuli would fail whenever the organ was moved in that plane, and all responses which the end organ subserves would appear to fail in that plane. Bodily deviations will always occur in a direction in which movement fails to provoke corrective afferent stimuli. All coordinated movement must be based on timely and imperceptible correction of deviations. If the three-dimensional labyrinth is entirely eliminated, the animal will deviate markedly, with a gross lack of correction, to the homolateral side. If only a part of the labyrinth is destroyed, the deviation will necessarily be in the plane of the part destroyed. This could still obtain if the canals were not integrated separately.

There are facts, however, which appear to make it a certainty that the canals have individual central representation; viz., if a canal is stimulated artificially, nystagmus is in the plane of the canal regardless of posture. Also in the rotation test, nystagmus is always parallel to the plane of rotation and hence parallel to the canal which lies in the plane of rotation. Now if the character of this nystagmus is not due to the control of the individual canal stimulated, it becomes necessary to postulate that some other influence is being brought to bear on the canal system, i. e., that of the utricle or that of the posture of the neck. The latter situation evidently does not obtain, because once the nystagmus is induced by rotation posture has no influence on its character. The evidence is in favor of individual and specific representation of the canals.

Relation Between the Canals and the Utricle: That the responses attributed to the canals can function in the absence of the utricles was demonstrated by McNally in the frog. (See section entitled "Utricle"). Likewise, de Kleijn and Magnus [7] and Wittmaack [8] demonstrated it with guinea pigs.

McNally and others demonstrated also that the responses attributed to the utricle can function in the absence of the canals.

There have been, however, experiments and clinical observations that pointed toward a certain interdependence and correlated function between the canals and the utricle.

De Kleijn and Magnus [7] and Wittmaack [8] discovered that the otoliths of guinea pigs could be displaced by centrifugation (2,000 revolutions per minute), while the cristae of the ampullae remained intact. In general the animals exhibited compensatory movements in all planes, but such movements were not sustained after rotation ceased. The dynamic functions remained normal, but the static functions were lost. Hence they concluded that the canals respond to movement while the otolithic organ is the source of reflexes of position. They stated that it would appear, as Maxwell had suggested, that the canals and the otolithic organ, though having separable functions, do reenforce each other and that as differentiation becomes more advanced the difference in function becomes more decided.

Camis [33] pointed out that if a rabbit is deprived of one labyrinth the vertical tonic deviation of the eye is maximal when the animal is lying on its side with the surviving labyrinth below. The postoperative nystagmus will be minimal when the animal is in this position. Conversely, when the intact labyrinth is uppermost the nystagmus is maximal and the deviation minimal.

Caloric Test.—There are certain facts about the caloric test that have proved intriguing and have challenged explanation since its dis-

covery, to wit, that cold and hot water cause the same phenomena but in opposite directions and also that the character of the nystagmus varies with the position of the head.

Bárány proposed an explanation of these reactions which has never been superseded by a more satisfactory one. He based his hypothesis on the supposition that convection currents are produced in the canals because of temperature gradients that result when cold or hot water is injected into the ear. Bárány proposed that in any given position the vertical canal is active. Cold water causes the endolymph to flow downward, and heat causes it to flow upward. On this thesis, he explained all the possible reactions obtained after douching the ear.

There are factors which speak against Bárány's theories that at least deserve consideration:

1. If the physical basis of stimulus to the canal is a current in the endolymph or its physiologic equivalent, such as is presumed to occur in rotation, then one may expect to see the effects of inertia of the endolymph, such as are seen after rotation. When rotation is stopped, at least a minute is required for the crista of the involved canal to become stabilized. The intensity of the nystagmus after douching is almost as great as that after rotation, and therefore the stimulating factor (displacement of the endolymph) must be almost as forceful. Yet, after douching, the character and plane of the nystagmus can be changed instantaneously by changing the position of the head. An acting canal can thus be immediately caused to cease acting, which is not true after rotation.

2. Bárány proposes that only a single canal is involved in the caloric test for a given position (i. e., the horizontal canal with the head back 60 degrees or forward 120 degrees), and yet the responses are almost as vigorous as those that result from the turning test, which involves the augmented or reciprocal functioning of paired canals. If rotation is done in a case of one dead labyrinth, the responses due to the remaining labyrinth are in the normal directions but are enfeebled, especially if there has not been time for adaptation to the loss of the labyrinth. Yet it is agreed that turning is a far more adequate stimulus and one to which the labyrinths are far more sensitive than temperature.

3. Nystagmus in only two planes—frontal or horizontal or a combination of the two—can be obtained by douching, but vertical nystagmus does not occur. The subjective sense of rotation, the past pointing and the falling also are limited to one plane—the frontal, or coronal. Whereas after turning the plane of nystagmus, which is the plane of reference for all other responses, remains fixed to the head when position is changed, the plane after douching remains always vertical to the floor (fig. 1).

EXPLANATION OF FIGURE 1

Fig. 1.—*A*, responses to rotation to the right with the head forward 120 degrees. (*a*) Position of head unchanged: Rotary nystagmus to the left (slow phase to the right), past pointing to the right, a tendency to turn to the right (hence no falling) and a sense of rotation to the left. (*b*) Position of head changed to 30 degrees forward (i. e., upright): Nystagmus unchanged, past pointing to the right with the arms forward, falling to the right and a sensation of falling to the left. (*c*) Position of head moved back to 60 degrees: Nystagmus unchanged, past pointing to the left, a tendency to rotate to the left (hence no falling) and a sensation of turning to the right.

B, responses to rotation to the right with the head inclined to the right shoulder. (*a*) Position of head unchanged: Vertical nystagmus downward (slow phase upward), past pointing to the right with the arms forward, a tendency to turn to the right (hence no falling) and a sensation of turning to the left. (*b*) Position of head changed to upright: Nystagmus unchanged, past pointing upward with the arms forward, falling backward and a sensation of falling forward.

NOTE.—By using this device it is easy to predict the normal reaction after turning. If one imagines a plane through the head and parallel to the ground when rotation is carried out and draws the arrows in the direction of rotation, the "after rotation" responses will be as follows: slow phase of nystagmus, past pointing and falling in the direction of the arrow, with a sensation of falling or turning opposite to the arrow. If now the position of the head is changed, the responses will maintain the same relationship to the plane and the arrow. The plane will remain fixed to the head.

C, responses to douching of the right ear with cold water with the head upright. (*a*) Position of head unchanged: Rotary nystagmus (in my experience seldom a pure rotary but usually a combined rotary and horizontal nystagmus) to the left (slow phase to the right), past pointing to the right with the arms forward, falling to the right and a sensation of falling to the left. (*b*) Position of head changed to 60 degrees backward: Horizontal nystagmus to the left (slow phase to the right), past pointing to the right with the arms forward, falling to the right and a sensation of falling to the left. (*c*) Position of head changed to 120 degrees forward: Horizontal nystagmus to the right (slow phase to the left) past pointing to the left with the arms forward, falling to the left and a sensation of falling to the right.

NOTE.—The device given for rotation does not hold for the caloric stimulus. When the head is placed 120 degrees forward there is a complete reversal of responses.

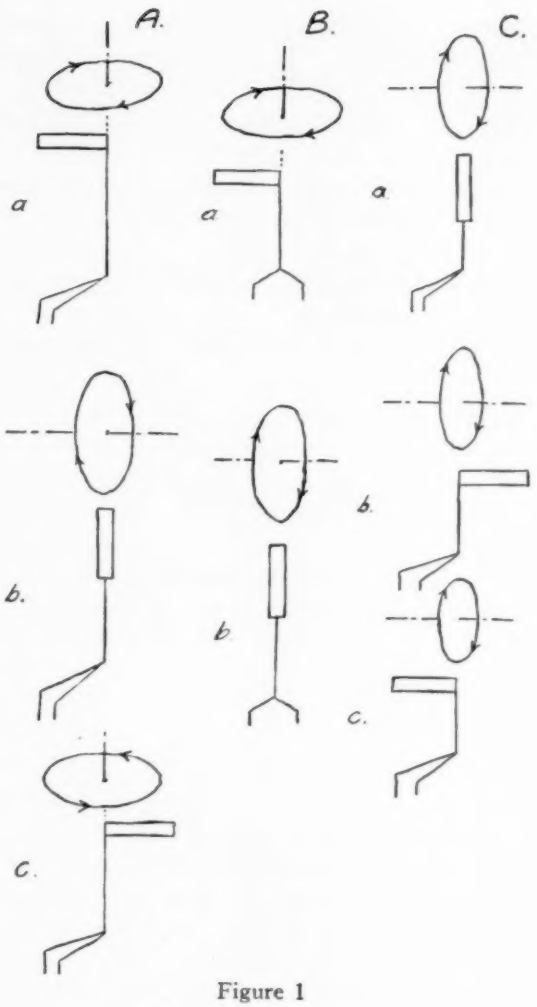

Figure 1

4. The physical mechanism hypothesized by Bárány for displacement of the endolymph has been given some attention, which on the whole has not brought forth confirmatory evidence.

Traina [73] on the basis of physical and mathematical considerations compared rotation and calorization in respect to the possibility and magnitude of movements of the endolymph. He concluded that in calorization movements of the endolymph would play a small role.

Kobrak,[74] after finding that a minimal stimulus of 5 cc. of water at a temperature of 5 or 10 degrees above that of the body could cause caloric responses, cast a great deal of doubt on Bárány's hypothesis.

Although it is obvious that the bony labyrinth can be cooled or warmed as a whole by douching, it is almost incredible that a temperature gradient could be produced in a canal sufficient to cause convection of the endolymph.

The transmission or conductivity of heat has been studied by Pinaroli (1912) on the human cadaver and by Schmaltz and Völger (1924) on both living and dead human bodies. Pinaroli found that when the temperature of the labyrinth was 14.5 C. and that of the douch water 40 C. the temperature of the region of the horizontal canal rose 0.5 degree in one minute and thirty seconds and 4 degrees in seven minutes. In the living subject the rise in temperature was smaller and slower.

The magnitude of the responses after calorization is almost as great as that of the responses after labyrinthectomy, which would lead one to suspect that the mechanisms have something in common. There is one outstanding difference in the nystagmus in the two cases. After labyrinthectomy the position of the head has no influence, while after calorization nystagmus varies with the position of the head. In seeking an explanation alternative to that of Bárány, one naturally looks first to the otolithic organ. Can it be possible that changes in temperature influence all three canals and that in such a case the otolithic organ governs the character of the nystagmus?

Relatively little work has been done to show the effects of calorization after elimination of the utricles. Evidence has been shown, however, that there is cooperation between the utricle and the canals. That is, in the absence of the otolithic organ deviation of the eyes is produced by rotation but is not maintained as it is in normal animals.

Chiloff [75] threw some light on the problem by demonstrating that changes in the position of the head definitely shorten the duration of

73. Traina, S.: Sul valore delle variazioni termiche dei canali semicircolari durante la prova, calorica, Valsalva **3**:454, 1927.

74. Kobrak, F.: Zur Wirgungsweise des kalorischen Schwachreizes, Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **18**:351, 1922.

75. Chiloff, C. Z.: De l'influence de l'irritation des otolithes sur les réflexes des canaux demicirculaires, Rev. de laryng. **48**:721, 1927.

nystagmus after rotation. For example, if rotation is carried out with the head forward and then the head is raised, the duration of the nystagmus is shorter than if the head is not raised. He found the same phenomenon to occur in cats and disproved a possible role of the reflexes of the neck by placing cats in plaster casts and obtaining the same results.

To show that the effect was due to the otolithic organ, he destroyed the otoliths in cats by centrifugation at the rate of 1,000 to 2,000 revolutions per minute, after the method of Wittmaack. Change in position of the head did not shorten the duration of nystagmus in these animals.

In a review of some theories, Chiloff [76] cited Mygind [77] as believing that caloric nystagmus is a function of both the otolithic organ and the canals and Borries as believing that while rotatory nystagmus is due to stimulation of the canals, caloric nystagmus is a function of the otolithic organ. Borries found pigeons in which he had destroyed the canals devoid of rotatory nystagmus, whereas caloric nystagmus could be obtained.

Chiloff pointed out again that de Kleijn and Lund had shown caloric nystagmus in animals in which the otolithic organs had been destroyed.

Mygind listed the following characteristics in distinguishing ampullary and otolithic nystagmus.

Nystagmus of canal origin:

1. Caused by sudden changes in position.
2. Disappears after several seconds.
3. Sudden appearance or onset.
4. Eye movements are always in the plane of head movement.
5. Depends on movement and not position of the head.

Nystagmus of otolith organ origin:

1. Appearance has no relation to rapidity of movement.
2. Position of head determines duration.
3. Requires a certain latent period to appear.
4. Depends on change in position of head and not movement.

It is true that Bárány's hypothesis would seem to fit most cases excellently. For example, when the head is back 60 degrees the horizontal canals are vertical, and lowering the temperature of the right one would be expected to cause a flow of endolymph downward or toward the ampulla. This current should cause horizontal nystagmus

76. Chiloff, C. Z.: De l'influence de l'irritation des otolithes sur les réflexes des canaux demicirculaires, Rev. de laryng. **48**:691, 1927.

77. Mygind, S. H.: How Does Caloric Nystagmus Arise? J. Laryng. & Otol. **40**:444, 1925; cited by Chiloff.[76]

to the left. Not only does this hold true, but when the head is placed 120 degrees forward the nystagmus reverses, as it theoretically should.

However, it might be argued that when the subject changes from supine to prone the utricle changes from a position of maximal to one of minimal stimulation, which could conceivably involve a change in the direction of the nystagmus. Such a theory would necessarily presuppose that changes in temperature constitute a stimulus to all the cristae, either depressant or excitatory, without eliminating the function of the utricle. The galvanic current and, of course, labyrinthectomy would be expected to overrule or eliminate the function of the utricle.

By referring to figure 1 *A* and *B*, it may be recalled that after rotation the plane of reference for all responses (which is parallel to the ground at the time of rotation) remains fixed to the head. Consequently the nystagmus does not change in character when the position of the head is changed. Past pointing and the falling reactions do change, however. Just how much they are influenced by the reflexes of the neck and by the otolithic organ is difficult to say. The effects induced by moving the head in the horizontal plane must be entirely due to the reflexes of the neck, because such a movement could not change the relation of the otoliths to gravity. However, when the head is moved in any other plane, the otolithic organs are stimulated and could therefore bring an influence to bear on the falling reactions and past pointing.

Now it will be noted that after douching (fig. 1 *C*) the plane of reference is not fixed to the head but always remains vertical to the ground. Consequently, when the position of the head is changed in the sagittal plane it is moved out of the plane of reference, and the character of the nystagmus will be altered according to the new relation of the head to the plane of reference.

It has been assumed that the utricle is *maximally* stimulated with the head back (supine position). In animals in this position the tone of the extensor muscles is at its greatest. This is in keeping with the observation that the tone of the extensor muscles is at its greatest in decerebrate rigidity. To obtain decerebrate rigidity, it is necessary that the primary vestibular nuclei be left intact. If they are removed decerebrate rigidity disappears. It might, therefore, he said that after cold calorization maximal stimulation of the utricle is associated with contralateral nystagmus, or the slow phase to the ipsilateral side, while minimal stimulation is associated with the reverse.

It should be recalled here that by the supine position for the cat is meant a position of the head far enough back (snout 0 degrees to 45 degrees to the horizontal) to place the macula horizontal, with the otolith hanging from it. The cat in this position is comparable with

the human being upside down. The cat in the reversed position (when the otoliths rest on the horizontal maculas) is comparable with the human being in the upright position. This position is associated with minimal extension in the decerebrate cat.

In the following consideration, therefore, of deflections of the otolith in one plane (fig. 2), "maximal" and "minimal" are used in a relative sense. When the head is moved from backward to forward, both utricles equally change from relative maximal to relative minimal stimulation. When the head is deviated to a shoulder, one utricle is maximally stimulated relative to the other.

If this reasoning is logical and the response dealt with is only from the vestibule that is being douched, one might expect when douching the right ear to get the same type of response with the subject's head back and with it held to the ipsilateral shoulder. Contrariwise, one

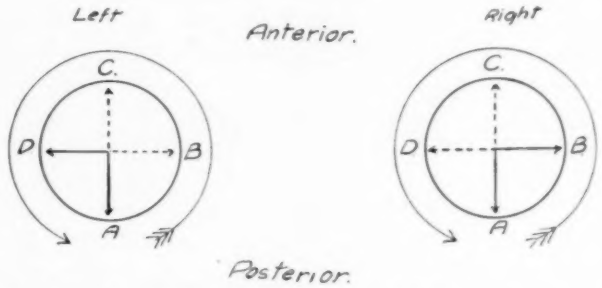

Fig. 2.—Probable plan of utricular function and influence on muscular tone.

This sketch is intended to represent a diagrammatic view of the utricular maculas, which in the normal upright position in man are nearly parallel to the ground. The solid arrows represent positions of relative maximal stimulation; the dotted arrows, position of relative minimal stimulation. If the head is considered to be gradually shifted so as to cause the otoliths to be shifted in all directions as one progresses in the direction of the large arrow, the following phenomena occur:

At *A*, supine position, the tone of the extensor muscles is relatively maximal and equal on the two sides.

At *B*, the right limbs are extended and abducted and the left flexed and adducted.

At *C*, prone position, the tone of the extensor muscles is relatively minimal on the two sides.

At *D*, the left limbs are extended and abducted, while the right limbs are flexed and adducted.

It is also highly probable that alterations in tone occur forward and backward (in the sagittal plane) as well as laterally (in the coronal plane).

might expect to get the same type of response when the subject's head is forward and when it is held to the contralateral shoulder. These expectations are justified because it has been shown that the supine

position induces maximal stimulation of both otoliths and is associated with maximal extension of the extremities on the two sides. Now, when the right ear is dependent, an unequal arrangement of the otoliths obtains. The right is maximally stimulated *relative* to the left, and the two otoliths in such positions function reciprocally (fig. 2). This view holds true in the caloric test; i. e., after the cold stimulus to the right ear the nystagmus is to the left with the head back or to the right shoulder and to the right with the head forward or to the left shoulder.[34]

Thus it is seen that two dimensional alterations in tone probably occur as the otoliths make a complete circle.

To elaborate further, the otolith may be deflected in two planes: (1) the vertical (push and pull effects) and (2) the horizontal (lateral deflections as indicated in figure 2). The tonal responses are in the first case purely flexion and extension. In the second case deflections of the body and its members are caused to occur in the coronal and the sagittal plane and in all planes between these two.

It is easy to see how, as the animal stands upright on its extremities, such tonal alterations can aid it in maintaining balance when it is subjected to changes in gravitational or centrifugal forces.

While Bárány's hypothesis fits all cases, the hypothesis of otolithic influence is tenable also.

Obviously the crucial test of the latter hypothesis would be to eliminate the otolithic organ and then to see if, after douching the ear, changes in the position of the head would bring about the changes in nystagmus that normally occur. So far as I can ascertain, this particular point has not been conclusively made.

Chiloff [76] stated, evidently basing the conclusion on experimental work, that the semicircular canals and the vesicles of the vestibule contribute simultaneously to caloric and postrotary nystagmus and to that produced by compression. However, I have not been able to find his protocols concerning caloric nystagmus especially in relation to changes in position of the head.

If the caloric responses are regarded as due to changes in intralabyrinthine tension, i. e., decreased tension after cold, as a result of contraction of the endolymph, and increased tension after heat, then the responses would be in keeping with those obtained by the fistula test. Suction is comparable to the cold stimulus and compression to the hot stimulus.

ROLE OF THE CEREBELLUM

Any one who undertakes to discuss the function of the cerebellum launches on an extremely involved and not too well understood subject. His treatment of it will necessarily be colored by his point of view. Before the role of the cerebellum in vestibular function is discussed, it is well to review the cerebellum in a more general way. A treatise

approaching completeness is obviously not within the scope of this paper, and the following discussion will therefore be a mere outline.

When the cerebellum is reviewed from evolutional, experimental and clinical standpoints, three properties stand out clearly: 1. It is a structure the office of which is to integrate impulses that have to do with motor function. 2. It is a supranuclear structure the removal of which does not paralyze the organism in the strict sense but markedly incapacitates motor function through incoordination. 3. There is no evidence that it subserves sensory function.

Histologic Connections.—The two types of connections, afferent and efferent, may be listed as follows:

Afferent Fibers:

1. Dorsal spinocerebellar tract (Flechsig) → inferior cerebellar peduncle → homolateral cerebellar cortex.

2. Ventral spinocerebellar tract (Gowers) → brachium conjunctivum → cerebellar cortex, partly crossed.

3. Gracile and cuneate nuclei (posterior columns) → inferior cerebellar peduncle → cortex of the vermis.

4. Olive → inferior cerebellar peduncle → cortex of cerebellum.

5. Medial, lateral and upper Bechterew nuclei (vestibular primary nuclei) → tractus nucleocerebellaris → partly to cortex of vermis, partly to nucleus emboliformis, nucleus globosus and nucleus fastigii.

6. Corticocerebellar tract [78] (frontal lobe, Arnold's bundle; temporal lobe, Türck's bundle) → middle cerebellar peduncle → cerebellar cortex.

Efferent Fibers: No efferent fibers lead directly from the cerebellar cortex to the pons, midbrain or cord except those from the anterior vermis to Deiters' nucleus. All others end in the cerebellar nuclei.[79]

1. Dentate, fastigial and contiguous nuclei → brachium conjunctivum → red nuclei.

2. Fastigial and contiguous nuclei → inferior cerebellar peduncle → substantia reticularis.

Experimental and Clinical Findings.—The literature concerned with this topic is voluminous but on the whole not highly controversial.

Experimental ablation is followed by a period of exaltation, during which certain dynamic phenomena appear.[80] These are strikingly

78. Winkelman, N. W., and Eckel, J. L.: Origin of the Cortico-Cerebellar System as Determined in Human Pathological Material, A. Research Nerv. & Ment. Dis., Proc. **6**:481, 1929.

79. Hohman, L. B.: The Efferent Connections of the Cerebellar Cortex: Investigations Based upon Experimental Extirpations in the Cat, A. Research Nerv. & Ment. Dis., Proc. **6**:445, 1929.

80. Thomas, A.: Cerebellar Functions, Nervous and Mental Disease Monograph 12, New York, Nervous and Mental Disease Publishing Company, 1912.

similar to those that occur after removal of the labyrinths. I feel that these dynamic or forced responses could be a result of a "release" of vestibular connections in the brain stem.

When the period of exaltation subsides, the animal exhibits the cerebellar deficiency complex referred to by the terms "asthenia," "atonia," "astasia," "asynergia" and "adiadokokinesis." The recent trend is to regard all the signs of cerebellar ataxia as manifestations of loss or impairment of postural tone. Walshe [81] classified the disorders in the following manner:

1. Abnormalities of resting muscle—loss of tone.
2. Abnormalities of muscular contraction.
3. Abnormalities of voluntary movement.
4. Effects of voluntary efforts at correction.

He aptly proposed that "it is solely voluntary movement, therefore, which is dependent upon cerebellar activity."

Holmes,[82] in an exhaustive study of cerebellar disorders in human beings, found signs in keeping with this view.

After some time the cerebellar disorders may be largely compensated. This compensation is evidently accounted for by the cerebral cortex, especially the frontal lobe. Aring and Fulton [83] showed that after removal of a cerebellar hemisphere and the contralateral premotor cortex (area 6 *A*) asynergia is extreme and is not compensated with time.

Large lesions of the cerebellum may be unassociated with abnormal signs, especially if the lesion is congenital or acquired in early life.[84]

I removed a dermoid cyst which had replaced an entire cerebellar hemisphere from an adult, who did not exhibit a single "cerebellar sign" before or after operation. She had presented increased intracranial tension, and the cyst was localized by ventriculographic examination.

Magnus and de Kleijn showed that righting reflexes persist after removal of the cerebellum, and Sherrington found the cerebellum unnecessary in the maintenance of the extreme tonus of decerebrate rigidity.[85]

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81. Walshe, F. M. R.: On the Disorders of Movement Resulting from Loss of Postural Tone, with Special Reference to Cerebellar Ataxy, Brain **44**:539, 1921.

82. Holmes, G.: The Symptoms of Acute Cerebellar Injuries Due to Gunshot Injuries, Brain **40**:461, 1917.

83. Aring, C. D., and Fulton, J. F.: Relation of Cerebrum to Cerebellum, Arch. Neurol. & Psychiat. **35**:439 (March) 1936.

84. Barker, L. F.: The Clinical Diagnosis of Internal Diseases, in Monographic Medicine, New York, D. Appleton and Company, 1916, vol. 4, p. 475.

85. Miller, F. R.: The Physiology of the Cerebellum, A. Research Nerv. & Ment. Dis., Proc. **6**:361, 1929.

The histologic connections of the cerebellum indicate that it is in connection with afferent stimuli having to do with the control of motor function. Proprioceptive stimuli, stimuli from the labyrinths and stimuli from parts of the cortex are integrated in the cerebellum, out of which a final expression emerges through the great efferent pathway.

The stimuli subserving muscular movements, tone and posture are not initiated sui generis in the cerebellum.

I do not believe that the cerebellum encompasses the only tracts subserving vertigo and past pointing which result from labyrinthine stimulation.

The cerebellum is an organ of equilibrium only in the purely reflex sense, being, as Sherrington has said, "the head ganglion of the proprioceptive system."

Relation of the Proprioceptors to the Cerebellum.—As soon as a muscle is contracted through the agency of the pyramidal system, stimuli are initiated in the proprioceptors. According to the evidence which has been deduced to date, the proprioceptors can be divided into two distinct classes. The impulses from one class are mediated in the posterior columns and probably in the two spinocerebellar tracts. These tracts lead to the cerebellum, and the posterior columns also lead to the thalamus. This class of proprioceptors contribute to the regulation of movement pattern and tonal distribution, and impulses from them are compounded in the cerebellum with labyrinthine and cortical impulses.

The other class of proprioceptors are initiative of the phenomenon that is called tone.

The "stretch" or "myotatic reflex" is, as Sherrington has said, "tone in the making" and requires an intact afferent and efferent nerve supply for its expression.

Deafferentation abolishes the phenomenon, and, as a converse of this, if the uncut peripheral efferent nerve is stimulated four or five times per second a continuous contraction curve is produced, while if the peripheral end of the cut nerve is stimulated separate twitches are evoked.[86]

While the basic neural arcs subserving the tone of both flexor and extensor muscles have successfully been shown to reside in the spinal cord proper, the tone of the extensor muscles predominantly requires intact *prespinal centers* for its basic expression.

Little has been proferred concerning the afferent tracts related to the tone of the extensor muscles, but the belief is practically uniform

86. Bayliss, W. M.: Principles of General Physiology, ed. 3, London, Longmans, Green & Company, 1920, p. 540.

that the efferent tracts are vestibulospinal and probably rubrospinal, reticulospinal and tectospinal. Probably afferent tracts are spinoolivary, spinotectal and—as yet unproved but possible—spinovestibular and spinorubral. At any rate, I believe the evidence indicates that the basic reflex arc determining the tone of the extensor muscles has its nuclei in the prespinal centers, while both afferent and efferent pathways reside in the anterolateral quadrants of the cord.

The abolition of decerebrate rigidity by section of the anterolateral columns and also the fact that the anterolateral columns are not indispensable to normal tone and movement pattern in man indicate that so far as the latter is concerned the posterior column–brain–pyramidal tract arc is of higher rank and has superseded almost entirely the ascending and descending extrapyramidal tracts.

The tone-producing mechanism appears to be independent of, separable from, but perfectly integrated with, movement pattern.[87] The control and distribution of tone is governed by efferent impulses from the cerebellum and striatum to the prespinal centers, chiefly the red nucleus and the reticular formation.

Thus it may be that the basic reflex arc determining the tone of the extensor muscles is not fully and completely released until the cerebellum is eliminated. At any rate, when it is released, as in decerebrate rigidity, the proprioceptive reflex which determines the tone initiates itself autonomously to a maximum degree, with the proprioceptors on the afferent side stimulated by contraction, while the resulting efferent stimuli maintain contraction. The cycle maintains itself with a striking lack of evidence of fatigue.[88]

At this point the concepts of decerebrate rigidity and its genesis must be reviewed.

Controversy has arisen chiefly on two scores: the role of the red nucleus in decerebrate rigidity and the role of the cerebellum.

Role of the Red Nucleus in Decerebrate Rigidity: That the red nucleus need or need not remain intact is upheld by the following evidence:

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87. I am preparing a report based on studies following sections of the anterolateral column, which I feel provides evidence for this view.

88. I do not believe it necessary to postulate a dual innervation to striated muscle to account for absence of fatigue. The evidence on muscular fatigue leaves the way open for the suggestion that fatigue as it is experienced may be largely a cortical phenomenon, the subjective sensation being referred to the periphery. Given a subcortical nonfatiguing neural mechanism, an intact muscle may be caused to maintain a state of spastic contraction indefinitely. The cortex is predominantly concerned with reflex sequences and not with repetitive or sustained discharges. Low level nuclear masses are capable of repeated discharge without evidence of fatigue.

Sherrington [36a] obtained the rigidity when he made the section at the caudal part of the diencephalon or at any level of the mesencephalon.

Thiele,[89] using cats and monkeys, concluded that the rigidity does not appear until the section passes through the caudal part of the thalamus. Successive slices increase rigidity until the trapezoid level is reached.

Ranson and Hinsey [90] pointed out that decerebration by the anemic method of Pollock and Davis [91] eliminates the function of the red nucleus and produces the most marked tonus of the extensor muscles. Rigidity is greater also if the section is made at the rostral border of the pons (red nucleus eliminated) than if it is made at the rostral border of the mesencephalon.

Ranson and Hinsey themselves [90] concluded that complete removal of the red nucleus is compatible with decerebrate rigidity of the highest grade.

Bazett and Penfield [92] obtained rigidity for more than two weeks with sections so low that only traces of the red nucleus were left and these probably were not functional. They stated that, though many reactions and postural reflexes disappear after the more caudal decerebrations, the essential character of the rigidity seems to be the same.

Rigidity persisted after median section of the midbrain, and hence section of the rubrospinal tracts at their decussation.

Miller [85] stated that considerable rigidity is developed if the plane of transection is made so as to leave the red nucleus intact.

These authors, while not denying that rigidity may be present if the red nucleus is left intact, are of the opinion that it is more complete if the red nucleus is eliminated.

Somewhat opposed to this view was the conclusion of Magnus [93] and of Rademaker [94] that sections above the level of the mesencephalon do not lead to decerebrate rigidity, since the mesencephalon contains centers essential for the maintenance of normal muscular tone.

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89. Thiele, F. H.: On the Efferent Relationship of the Optic Thalamus and Deiters Nucleus to the Spinal Cord with Special Reference to the Influx Theory of Dr. Hughlings Jackson and the Genesis of the Decerebrate Rigidity of Ord and Sherrington, J. Physiol. **32**:358, 1905.

90. Ranson, S. W., and Hinsey, J. C.: Extensor Tonus After Transection of the Brain Stem at Varying Levels, J. Nerv. & Ment. Dis. **70**:584, 1929.

91. Pollock, L. J., and Davis, L. E.: Studies in Decerebration: I. A Method of Decerebration, Arch. Neurol. & Psychiat. **10**:391 (Oct.) 1923.

92. Bazett, H. A., and Penfield, W. G.: A Study of the Sherrington Decerebrate Animal in the Chronic as Well as the Acute Condition, Brain **45**:185, 1922.

93. Magnus, R.: Körperstellung, Berlin, Julius Springer, 1924.

94. Rademaker, G. G. J.: Die Bedeutung der roten Kerne und des übrigen Mittelhirns für Muskeltonus, Körperstellung und Labyrinthreflexe, Berlin, Julius Springer, 1926.

Rademaker stated that the red nucleus is the center which presides over normal distribution of tonus and that normal tonus still persists after removal of the cerebral hemispheres, corpus striatum, optic thalami and corpora quadrigemina. Then, however, removal of the red nucleus, section of the rubrospinal tracts or section of Forel's decussation will cause tonus of the extensor muscles.

Role of the Cerebellum in Decerebrate Rigidity: The experiments of Weed [95] would seem to indicate at first that decerebellation abolishes decerebrate rigidity. In one cat, however, this was not true. Also, removal of the cerebellum in one cat resulted in extensor rigidity in all limbs. This rigidity disappeared in time, and after four weeks it reappeared after decerebration.

It would seem that in experiments of this kind, in which massive mutilations of the brain necessarily cause a great deal of shock and exhaustion to the animal, positive results are more dependable than negative; i. e., if in one animal decerebellation does not abolish decerebrate rigidity, a conclusion based on this result is more likely to be correct than one based on contrary results in many other animals.

At any rate, the results of Sherrington,[96] Horsley,[97] Thiele [89] and Magnus [98] leave no doubt that decerebellation does not abolish decerebrate rigidity. In fact, it has appeared that decerebellation has occasionally, if anything, increased the rigidity.

Effects of elimination of Deiters' nuclei and vestibular spinal tracts. The works of Lorente de Nó,[99] Papez [100] and Gray [101] demonstrated that an operation destroying the vestibulospinal tracts (chiefly by sagittal section) and leaving the red nucleus with the rubrospinal tracts intact does not completely abolish decerebrate rigidity.

Bernis and Spiegel [102] destroyed Deiters' nuclei on both sides without producing complete loss of rigidity.

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95. Weed, L. H.: Observations upon Decerebrate Rigidity, J. Physiol. **48**: 205, 1914.

96. Sherrington, C. S.: Decerebrate Rigidity and Reflex Coordination of Movements, J. Physiol. **22**:319, 1898.

97. Horsley, V.: The Functions of the Cerebellum, Brain **29**:446, 1906.

98. Magnus, R.: Welche Teile des Zentralnervensystems müssen für das Zustandekommen der tonischen Hals- und Labyrinthreflexe auf die Körpermuskulatur vorhanden sein? Arch. f. d. ges. Physiol. **159**:224, 1914.

99. Lorente de Nó, R.: Die Labyrinthreflexe auf die Augenmuskeln nach einseitiger Labyrinthexstirpation, Berlin, Urban & Schwarzenberg, 1928.

100. Papez, cited by Ranson and Hinsey.[90]

101. Gray, L. P.: Some Experimental Evidence of the Connections of the Vestibular Mechanism in the Cat, J. Comp. Neurol. **41**:319, 1926.

102. Bernis, W. J., and Spiegel, E. A.: Die Zentren der statischen Innervation und ihre Beeinflussing durch Klein- und Grosshirn, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **27**:197, 1925.

The agreement is general that transection of the brain stem caudal to the vestibular nuclei abolishes decerebrate rigidity completely.

The following conclusions seem tenable:

1. Decerebrate rigidity can be obtained only with the afferent and efferent peripheral nerves intact.

2. The red nucleus may or may not be intact, but the section must be no further rostrad than the rostral border of the red nucleus.

Indeed, from the vast work on this subject one gains the impression that, though the preferred point of section has oscillated rostrad and caudad to the red nucleus, an agreement has finally been reached that the most pronounced rigidity is evoked by section through the nucleus. This suggests that disconnection of the major cerebellar efferent fibers is necessary to provide optimum decerebrate rigidity. In other words, the release of the reflex arcs responsible for the maintenance of decerebrate rigidity is not complete without decerebellation.

Sherrington [103] showed that section of the ventrolateral column abolishes decerebrate rigidity ipsilateral to the section and caudal to it.

The destruction of either reflex arc alone (vestibular or rubral) does not completely abolish the rigidity.

Effects of stimulation of the cerebellum in decerebrate rigidity. The following effects can be obtained only with the red nucleus intact.

There is general agreement that the rigidity of decerebration may be inhibited by stimulation of the anterior cortex of the vermis (Weed; [95] Sherrington; [104] Lowenthal and Horsley; [105] Cobb, Bailey and Holtz [106]). The last three authors by stimulating a superior cerebellar peduncle obtained ipsilateral inhibition of rigidity.

Miller [85] described various postures that result from stimulation of the various nuclei of the cerebellum.

The results of cerebellar stimulation are not out of keeping with my expressed views. Stimulation of the great efferent system of the cerebellum roughly and momentarily replaces the influence of the cerebrum, which has been disconnected. Decerebellation on the other hand completes the release of the vestibular and the rubral arc in the decerebrate preparation.

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103. Sherrington, C. S.: Flexion-Reflex of the Limb Crossed Extension-Reflex, and Reflex Stepping and Standing, J. Physiol. **40**:28, 1910.

104. Sherrington, C. S.: On Reciprocal Innervation of Antagonistic Muscles, Proc. Roy. Soc., London **60**:414, 1896-1897.

105. Lowenthal, M., and Horsley, V.: On the Relations Between the Cerebellar and Other Centers (Namely Cerebral and Spinal) with Especial Reference to the Action of Antagonistic Muscles, Proc. Roy. Soc., London **61**:20, 1897.

106. Cobb, S.; Bailey, A. A., and Holtz, P. R.: On the Genesis and Inhibition of Extensor Rigidity, Am. J. Physiol. **44**:239, 1917.

I believe that the attitudes and postures which are struck when the great efferent complex is stimulated are chance motor schemata that result from the dominant stimulus of this or that group of neurons. The response to this unphysiologic stimulus must be as heterogeneous in relation to the cerebellum as the movement of an extremity in relation to the cerebral cortex when a peripheral nerve is stimulated. The chance attitudes sometimes express inhibition and sometimes excitation. They seldom are reproduced exactly, although the stimulus applied may be thought to be the same. Such induced responses are probably simulacra of postures struck by the normal animal, as if his motion picture were stopped on a single frame.

Relation of the Cortex to the Cerebellum.—It would be almost expected that with the development of the neopallium, in which is elaborated volitional patterns, provision for direct association with the cerebellum would be made. Such an association would provide a mechanism by which the very visualization or cortical elaboration of a movement could be conditioned to its pattern in the cerebellum. A double mechanism would thus be provided for rapid, smooth performance—the proprioceptive reflex and the corticocerebellar reflex working concomitantly and in harmony.

To strike an example, the visualization of speech and the execution of its reflex sequence must be elaborated in the cortex. The muscles of speech, to be sure, would function and their tonal alterations and movement patterns be controlled through the proprioceptive-cerebellar reflexes. However, a smoother and more substantial function would be expected if such patterns were constructed or even reenforced in the cerebellum through association with the cerebral cortex.

I should like to recall what I have termed the "prepared" or cortically evoked posture (section entitled "Extralabyrinthine Reflexes Concerned with Equilibrium, Stabilization of the Visual Fields and Posture"). The effects of unexpectedly stepping off a curb or being suddenly pushed from behind are common experience. Although proprioceptive and labyrinthine stimuli and reflexes are evoked, they are not sufficient for meeting the emergency gracefully. The recognition of what is to come may be conveyed by any sensory channel—not necessarily through vision. Undoubtedly the corticocerebellar association plays an important role here.

Relation of the Vestibular Labyrinth to the Cerebellum.—The evidence indicates that dynamic and statotonic influences of the labyrinth can function through pathways in the brain stem exclusive of the cerebellum. I am not convinced that the pathways mediating vertigo and past pointing course only through the vermis of the cerebellum. I have removed a solid astrocytoma from the vermis in 2

cases in which there was almost total destruction of the vermis and certainly injury to the roof nuclei. The labyrinthine functional tests were normally performed with respect to past pointing and the presence of vertigo, before and after the operation.

Other evidence supporting this view is the fact that decerebellate pigeons will in time recover much of their capacity for flying, whereas labyrinthectomized pigeons are permanently crippled. The same contrast may be drawn in reference to incoordination after the two experiments in other animals.

However, there is a definite cerebellar counterpart, and the labyrinthine stimuli are here integrated with the proprioceptive stimuli. Just to what exent, if at all, the labyrinth can induce tone in the sense of causing a contraction of muscle is difficult to say. As brought out before, the evidence would seem to indicate that the major function of the labyrinth is to alter or modify the tone that is occasioned by the proprioceptive reflexes. Thus, when eliciting postural changes (Magnus-de Kleijn reflexes) in cases of decerebrate rigidity, one is undoubtedly viewing the effects of tonal distribution in pure form superimposed on a released extensor rigidity mechanism.

These movements are delayed and sluggish, which would indicate that the afferent impulses concerned do not ultimately cause an active contraction of muscle but alter the state of excitability of the prespinal centers in such a way (pattern) that the taut muscles stretch their relaxing antagonists to reach a new equilibrium. This does not mean that tonal reflexes are normally slow of change. In voluntary movement their changes must be rapid and concomitant with the movement to be purposive.

It would appear that all afferent stimuli ultimately governing tone, such as those from the labyrinths, "set the switchboard," as it were, so that the voluntary contraction will be accompanied by the proper degree of its reenforcing tone.

Consequently the influence of the labyrinths on tone is not exhibited unless the proprioceptors are functioning concomitantly. However, the influence of the labyrinth on the extraocular muscles during narcosis should not be overlooked nor should the fact that the labyrinth can evoke some change in tone in an extremity deprived of afferent connections.

Therefore, while the cerebellum is not indispensable to the independent expression of any of the three great afferent complexes considered (proprioceptive, cortical and labyrinthine), it is nevertheless indispensable to their compounding and total integration. It functions, therefore, as any other reflex system, and its office is fully expressed only in the event of volitional movement.

BLOOD SEDIMENTATION IN ACUTE OTITIS MEDIA AND ITS COMPLICATIONS

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In contrast to the voluminous literature found on the subject of sedimentation rates in other branches of medicine there is a noticeable scarcity of literature on this subject in the field of otolaryngology. Hart,[1] from a series of 25 cases of acute otitis media and its complications in which sedimentation studies were made and cultures of the causative organism taken, concluded that in the main the sedimentation rate is proportional to the severity of the infection, regardless of the nature of the complication. He stated the belief that this work offers no help in the differentiation of otitis media from its complications. He did, however, express the opinion that it is of definite prognostic value. Weiss [2] concluded from a series of cases of chronic otitis media that the sedimentation rate in this condition is normal. However, a high or rising rate is suggestive of impending mastoiditis. He declared himself in accord with other workers in the belief that this test is of value in following the course of otitis media. Lintz,[3] supporting the observations of Weiss that chronic infections do not affect the sedimentation rate, concluded from a series of 30 cases of chronic sinusitis that there is no significant change in the rate of sedimentation. In 13 cases of chronic tonsillitis the results were essentially as stated.

Despite the great amount of experimental work done on this subject it has not been accepted by the clinician as having a practical value. This is due in all probability to the mass of nonessential technical detail befogging the fundamental simplicity of the procedure. Walton [4] described the few factors one had to bear in mind. These are:

1. The sedimentation tube should be of a size sufficient to prevent too early "packing" of the red blood cells.

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† Dr. Hume died on Oct. 29, 1938.

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1. Hart, V. K.: Sedimentation Rate in Acute Otitis Media and Its Complications, Arch. Otolaryng. **9**:622-624 (June) 1929.
2. Weiss, A.: Laryngoscope **38**:85-93 (Feb.) 1928.
3. Lintz, R. M.: J. Lab. & Clin. Med. **21**:1259-1264 (Sept.) 1936.
4. Walton, A. C. R.: J. Lab. & Clin. Med. **18**:711-723 (April) 1933.

2. The temperature should range between 19 and 23 C.

3. The tube should be maintained in an upright position, as deviation from the perpendicular markedly affects the rate of sedimentation.

4. The anticoagulant used should be in a concentration such as to be isotonic with blood.

Much ado has been made in the literature over what effect anemia has on the rate of sedimentation. It is our belief that the view taken by Bannick and his co-workers [5] is the most rational. It is their contention that sedimentation rates are of value only when variations from the normal are of significant amount. For red blood counts of 4,000,000 or over the correction factor is so small as not to affect the final result appreciably. For counts under that figure they advocate the correction factor described by Rourke and Ernstene.[6] In other words, a determination of the sedimentation rate should be interpreted as a qualitative indicator of activity.

In the past, it is of interest to note, in studies on the sedimentation rates in otitis media there has been no attempt to follow the case through to resolution or complication. It is the purpose of this paper to show the trend of the rate of sedimentation in otitis media and its complication by repeated estimations and along with this to bring out any relation that may exist between the white blood count and the sedimentation rate. In addition, it is hoped to demonstrate the ease with which this simple laboratory procedure may be incorporated into the usual office routine.

There are a number of different technics in the literature, each with its own adherents. In this country the principal tests used are those of Linzenmeier, Cutler and Westergren. As it is not the purpose of this paper to discuss the advantages and disadvantages of the various procedures, we shall omit the highly technical discussion involved. However, as it is in part the purpose of this work to demonstrate the ease with which determinations of the rate of sedimentation may be carried out in the office or laboratory, it is thought advisable briefly to justify the choice of the Westergren pipet, which was used. It is of such a size as to fulfil the requirements discussed by Walton; only one reading is necessary, and only 1 cc. of blood is required. This is an obvious advantage if repeated readings are to be taken on the same patient.

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5. Bannick, E. G.; Gregg, R. D., and Guernsey, C. M.: Erythrocyte Sedimentation Rate: Adequacy of Simple Test and Its Practical Application in Clinical Medicine, J. A. M. A. **109**:1257-1265 (Oct. 16) 1937.

6. Rourke, M. D., and Ernstene, A. C.: J. Clin. Investigation **8**:545-559 (June) 1930.

MATERIALS AND PROCEDURE

The materials used are:

1. A block of wood 2 by 4 by 6 inches (5 by 10 by 15 cm.), containing three holes of a size just to contain a pipet in an upright position.
2. A 3.8 per cent solution of sodium citrate in a vaccine bottle fitted with a rubber diaphragm.
3. A 1 cc. tuberculin syringe.
4. A small cork plug cut to fit into the lower end of the pipet.

Procedure.—One-tenth cubic centimeter of the sodium citrate solution is drawn into the syringe, which is tilted a number of times to moisten the entire barrel with the anticoagulant. The patient's arm is prepared for venipuncture, and 1 cc. of blood is withdrawn (last 0.1 cc. is estimated). A few air bubbles are aspirated into the syringe and the latter rocked in order thoroughly to mix the anticoagulant and blood. Then, after all air bubbles are expelled, the needle, with the syringe attached, is inserted into the lower end of the pipet for a distance of about ¼ inch (0.6 cm.) and the blood slowly forced into the tube, the lower end of which should be tilted upward slightly. When the zero mark at the distal end of the tube is reached, the little cork plug is inserted into the lower end and the pipet into the wooden block. The time is noted, and at the end of an hour the fall of the red blood cells is read directly off the calibrated tube. This figure is the sedimentation rate, and for this tube the upper normal is a fall of 20 mm. in one hour. At the same time a complete blood picture is taken. This should include a count of the young forms. If the erythrocyte count is 4,000,000 or over, no correction for anemia is necessary. If, on the other hand, it is under that figure, correction is made according to the method of Rourke and Ernstene. In the series to follow no correction was necessary.

REPORT OF CASES

CASE 1.—B. C., a white girl aged 11 years, was first seen on Feb. 3, 1938. The history and findings were those of acute otitis media following in the course of acute infection of the upper part of the respiratory tract. There was no history of disease of the ear, nose or throat.

Summary of Case.—This case was one of an uncomplicated acute catarrhal process. The blood picture was that of a septic process of moderate degree, with a slight increase in the number of young forms. The sedimentation rate was well within normal limits. Eleven days later, paralleling the clinical regression, the blood count was within normal limits, though the number of young forms had not changed. The sedimentation rate was approximately half that found in the acute process.

CASE 2.—E. C., a Negro woman aged 27, was first seen on Feb. 5, 1938. The history and findings were those of acute catarrhal otitis media, with a serous effusion. There was no history of disease of the ear, nose and throat.

Summary of Case.—In this case there was presented the picture of acute serous catarrhal otitis media that had gone on to suppuration and then resolution. In the serous stage, seen on February 5, the blood picture and sedimentation rate duplicated their performance in case 1. The blood picture was that of a moderate septic process; the sedimentation rate was slightly above normal. On February 7, two days later, the process had changed from serous to purulent otitis media,

with the pus under pressure, as shown by the bulging of the tympanum. There was an increase in the white blood count, with a continuation of the young forms. The rise in the sedimentation rate was marked. Tympanotomy was refused at this time. Two days later, on February 9, there was clinically a regression of

Laboratory Data

Diagnosis	Date	White Blood Cells	Polymorphonuclears (%)	Myelocytes (%)	Juvenile Cells (%)	Stabs (%)	Sedimentation Rate, Mm. per Hr.
1. Acute catarrhal otitis media	2/ 3/38	12,000	55	2	0	2	13
	2/14/38	5,000	37	1	1	2	6
2. Acute serous otitis media becoming purulent otitis	2/ 5/38	11,000	75	2	4	6	22
	2/ 7/38	17,000	89	3	1	5	48
	2/ 9/38	6,000	79	0	1	1	75
	2/12/38	5,000	61	2	3	1	57
3. Acute catarrhal otitis media becoming purulent otitis	2/14/38	15,000	72	3	6	7	19
	2/19/38	11,500	77	4	4	6	82
4. Exacerbation of chronic otitis media	2/16/38	6,000	59	0	11	0	14
	2/19/38	10,000	58	1	2	3	9
5. Pneumococcic mastoiditis....	2/ 7/38	6,000	38	0	0	0	58
6. Exacerbation of chronic mastoiditis	1/26/38	10,000	34	1	1	1	40
	1/29/38	8,000	36	1	3	2	24
	2/ 4/38	12,500	52	1	3	5	35
	2/12/38	6,000	58	0	2	3	11
7. Acute mastoiditis.....	1/25/38	15,000	65	2	0	11	80
	1/28/38	23,000	58	1	0	0	91
	2/ 2/38	15,000	67	2	2	5	78
8. Acute mastoiditis.....	3/ 5/38	8,500	71	3	2	3	80
	3/ 6/38	.....	..	..	..	..	67
	3/ 7/38	.....	..	..	..	..	101
	3/ 8/38	7,000	83	4	7	7	115
	3/ 9/38	9,500	72	5	4	7	120
	3/11/38	7,000	75	3	3	8	118
	3/12/38	.....	..	..	..	..	119
	3/14/38	6,000	46	4	4	2	97
	3/16/38	5,500	40	4	3	6	93
	3/18/38	8,000	58	0	2	2	80
	3/23/38	.....	..	..	..	..	57
	3/26/38	.....	..	..	..	..	65
	3/27/38	.....	..	..	..	..	56
	4/11/38	4,000	53	0	3	5	34
9. Acute mastoiditis with external rupture	3/16/38	12,000	81	1	0	3	126
	3/18/38	14,000	74	2	6	3	132
	3/22/38	12,000	73	3	4	3	136
	3/25/38	10,500	54	3	4	2	126
	3/30/38	9,000	58	0	1	0	124
10. Exacerbation of chronic mastoiditis	3/ 8/38	5,000	60	1	1	4	34
	3/ 9/38	5,500	67	2	5	5	33
	3/10/38	.....	..	..	..	..	30
	3/11/38	5,000	64	3	5	5	26
	3/14/38	5,000	37	4	2	3	24
	3/16/38	5,000	56	2	0	2	30
	3/22/38	6,500	64	2	3	1	47
	3/25/38	6,500	74	2	3	6	50
	3/30/38	.....	..	..	..	..	41
11. Exacerbation of chronic mastoiditis	3/16/38	8,000	62	2	1	2	14
	3/26/38	9,500	87	4	5	5	31
	3/30/38	10,500	70	2	0	5	31

symptoms and findings. At the same time the differential count dropped to normal, with a decrease in the young forms. The sedimentation rate, however, was markedly increased. Three days later, February 12, with subsidence of all clinical symptoms, the white blood count dropped to normal limits, though the number of young forms had increased. The sedimentation rate had dropped, though still above normal; this observation was in keeping with the residual thickening of the tympanum and the moderately impaired mobility. Contact with the patient was lost at this time.

CASE 3.—A. O., a white man aged 28, was first seen on Feb. 14, 1938. The case was one of acute catarrhal otitis media, becoming purulent. There was no history of disease of the ear, nose or throat.

Summary of Case.—This patient when first seen presented the clinical and laboratory findings seen in case 2. Two days later, on February 16, the tympanum was "beefy red" and bulging in the posterior superior quadrant. A tympanotomy was done, releasing thick pus under pressure. At this time there was tenderness over the mastoid tip, probably due to periosteal congestion. Three days later there was no change in the clinical findings. Both the blood picture and the sedimentation rate showed an increase in the septic process. The patient at this time did not return for further treatment. So far as this case was observed we found it to parallel case 2.

CASE 4.—T. C., a white woman aged 37, was first seen on Feb. 16, 1938. The case was one of acute exacerbation of chronic otitis media. The patient had had an intermittently discharging ear for two years. The further history was irrelevant. Examination showed a perforation of the inferior margin, with a thick creamy pus under moderate pressure. Roentgenograms showed a well pneumatized mastoid, slightly cloudy.

Summary of Case.—Here was presented a new type of condition: chronic otitis media, with hyperplasia and fibrosis of the mucosa. We were impressed by the delayed response of the white blood cells to the time when clinically the acute process had subsided, February 19. The sedimentation rate performed as in case 1.

CASE 5.—M. G., a Negro woman aged 41, was first seen on Feb. 7, 1938. The case was one of probable pneumococcic mastoiditis. However, as the patient did not submit to surgical intervention, the final diagnosis will have to be speculative. There was no history of disease of the ear, nose or throat. The history given was that of occasional "sticky" pains in the left ear, associated with a feeling of fulness. Determination of loss of hearing by tuning forks showed a marked raising of the low tones. The high tones were maintained. Examination revealed a thickened drum of a grayish brown leathery appearance. There was moderate retraction. No perforation was seen. Roentgenograms showed a large mastoid of the pneumatic type, with clouding and destruction of cells in the region of the antrum and tegmen. Unfortunately, after the first examination the patient did not return.

Summary of Case.—It is believed that in view of the few symptoms offered, the marked loss of hearing, the history of chronicity and the objective findings in the ear that this was a case of type III pneumococcic mastoiditis. The roentgen findings bear this out. Of significance is the lack of leukocytic response to this destructive process. To be noted is the elevated sedimentation rate.

CASE 6.—J. B., a white boy aged 5 years, was first seen on Jan. 2, 1938. The case was one of acute exacerbation of chronic mastoiditis. The history revealed intermittent episodes of "running ear" since the patient was 6 months of age and pain and discharge of thick yellow pus of three days' duration. Examination showed a posterior marginal perforation, with the discharge pulsating. There was also definite tenderness over the tip of the mastoid. Roentgenograms showed an acellular sclerotic mastoid.

Summary of Case.—This case was similar to case 5, though it may be assumed to have carried the pathologic picture one step further, that is, to the stage of

a chronic sclerotic mastoid with its associated chronic otitis media. Here we found the blood picture and sedimentation rate in close agreement. Despite the initial severity of the clinical picture, we found the laboratory data indicating a return to normal in approximately sixteen days. This observation agreed with the clinical findings. It is apparent that the pathologic picture in this type of case favors either early resolution or complication. Furthermore, the laboratory data do not parallel the clinical severity.

CASE 7.—A. S., a white boy aged 5 years, was first seen on Jan. 25, 1938. The case was one of acute mastoiditis. The history revealed severe pain in the right ear and increase in the aural discharge of three days' duration and pain in the involved ear which had developed on Dec. 28, 1937. Three days after the onset tympanotomy had been performed, with relief of pain. The discharge, however, had continued until the onset of the symptoms described. Examination showed marked tenderness over the antral area of the mastoid region, periosteal thickening over the mastoid process and thickening of the drum, with perforation of the attic. Pus was seen in the latter under pressure. Roentgenograms showed an acellular sclerotic mastoid, presenting some softening of the shadow in the antral region. The patient was operated on on Jan. 27, 1938, and, contrary to the roentgen findings, a well pneumatized mastoid was found, with the periantral cells destroyed. Free pus was found in this location.

Summary of Case.—In this case there was an active response of the blood picture and the sedimentation rate, quite in keeping with the clinical picture. On January 28, one day after surgical intervention, there was an increase in both. This was in all probability a response to the destruction of tissue during surgical intervention. Unfortunately we were unable to continue this series, as acute bronchitis developed on February 3 and it was thought best to discontinue the laboratory work.

CASE 8.—E. G., a white man, was first seen on March 5, 1938. The case was one of acute mastoiditis. The patient entered the hospital with the complaints of persistent parietal headache and a discharging ear on the same side. He stated that about a month before entrance into the hospital he was taken ill with the "flu." On the third day of his illness an aching developed in the left ear. On the second day after the onset, the drum opened spontaneously to discharge a thick creamy pus. This discharge had continued until the time of his admission. About two weeks prior to entrance persistent parietal (left) headache had developed. This was more severe at night. Examination revealed marked mastoid tenderness and periosteal induration. Marked sagging of the posterior superior wall of the canal was noted. This was of such extent as to prevent a view of the tympanum. Roentgenograms showed complete destruction of a well pneumatized mastoid. The patient had a simple mastoidectomy on the day of admission, March 5. The pathologic picture revealed by surgical intervention was that of coalescent mastoiditis, with necrosis of the sinus plate and tegmen. The sinus and dura presented a gray appearance and were covered with granulations.

Summary of Case.—In this case there was a repetition of facts already noted. On admission there was a relatively high sedimentation rate, associated with a blood count in keeping with the clinical findings. After surgical intervention the patient had a severe chill, with a rise in temperature to 103 F. On the second postoperative day there was a marked elevation in the sedimentation rate. From March 6 to 9 the patient had a moderately septic temperature. The further post-

operative course was uneventful. Ten days after the initial operation a secondary closure was performed, and the patient was discharged on April 11. Of note in this case is the slow return to normal of the sedimentation rate as compared with the relatively rapid return to normal of the blood picture.

CASE 9.—E. N., a white woman aged 45, was first seen on March 16, 1938. The case was one of acute mastoiditis, with rupture of the external mastoid cortex. The patient entered the hospital with the complaint of pain and swelling behind the left ear of three days' duration. The history was that of the usual acute otitis media that goes on to acute mastoiditis. This exception may be noted. About one month before admission a mastoidectomy had been advised and was refused. Examination revealed a large postauricular fluctuant inflammatory mass. There was marked tenderness over the mastoid process. The tympanum was thickened and injected. Pus was seen to pulsate through a posterior superior perforation. There was some suggestive sagging of the posterior superior wall of the canal. No roentgenogram was taken. At operation approximately 2 ounces (59 cc.) of pus welled up into the incision. With removal of the remaining mastoid cortex the site of rupture was found to be around the exit of the mastoid emmissary vein. Further pathologic changes were those of coalescent mastoiditis, with necrosis of the sinus plate and tegmen. The dura and sinus were covered with friable granulations. The postoperative course was uneventful.

Summary of Case.—This case demonstrated only one new point, the effect of massive destruction of tissue on the rate of sedimentation. There is found the usual postoperative rise in the rate and then a slow drop to normal. This is of significance in view of the normal differential count. The further course of this case will be reported later.

CASE 10.—M. P., a white woman aged 61, was first seen on March 8, 1938. The case was one of acute exacerbation of chronic mastoiditis. The patient entered the hospital complaining that she had had pain in the right ear for about three weeks. With this there was a foul discharge. The history is that of an intermittently discharging ear, with a progressive loss of hearing of the affected ear. Examination showed a thickened tympanum, practically destroyed. The malleus was absent. The discharge had a foul odor. Roentgenograms showed a sclerotic acellular type of mastoid. At operation, on March 15, a sclerotic mastoid was found. There was a cholesteatomatous mass in the antrum and the periantral regions. The incus, like the malleus, was absent. A radical mastoidectomy was performed. The postoperative course was uneventful.

Summary of Case.—This case presents a new picture, that of a chronic pathologic process. The laboratory findings are noteworthy in that the values are all low, though still within the pathologic range. Of further interest is the fact that their return to normal limits is rapid as compared with that in other cases.

CASE 11.—H. H., a white boy aged 14, was first seen on March 16, 1938. The case was one of acute exacerbation of chronic mastoiditis and paralleled case 10 so closely that we will omit the description and discussion of it other than to say that the patient was operated on on March 21. A radical mastoidectomy was performed, and the postoperative course was uneventful. The laboratory data, as far as we were able to gather them, were essentially similar to those in case 10.

SUMMARY

Unfortunately, definite conclusions cannot be drawn from this work, because of the few cases observed and at times the rather incomplete data obtained. However, despite these factors, there was at least 1 representative of each of the several types of pathologic picture found in the middle ear and mastoid.

In the case of simple uncomplicated acute otitis media the rate of sedimentation remained well within normal limits, despite a comprehensive shift in the blood picture to that of a septic process. Of interest here is the observation that when the serous effusion of acute otitis media becomes purulent there is a prompt rise in the sedimentation rate. The blood picture in these cases is not distinctive, as it points only to an acute process. A point to be noted here is the slow return of the sedimentation rate to normal as compared to the relatively rapid change of the blood picture. Apparently the sedimentation rate follows the clinical regression of the pathologic changes in the middle ear.

In the cases of chronic otitis media with acute exacerbations neither the blood picture nor the sedimentation rate was appreciably changed. This might be explained by the fibrosis of the tympanum and mucosa of the middle ear and mastoid process. With fibrosis there is a decrease in vascularity, and with the latter there is less absorption of toxins. In explaining the lack of significant change in the rate of sedimentation in uncomplicated acute otitis media, one might assume it to be due to the fact that despite the marked congestion of the involved parts there is no actual destruction of tissue. This explanation would seem to indicate that changes in the rate of sedimentation are dependent on the destruction of tissue, an idea which is further borne out in the case of probable pneumococcic (type III) mastoiditis. Despite the essentially normal blood picture there was an elevated sedimentation rate.

In case 6, presented as a case of acute exacerbation of chronic mastoiditis, there was a rise in the sedimentation rate, associated with a blood picture indicative of infection. Here there was reason to believe a low grade destructive process was going on. On conservative treatment resolution occurred, and the sedimentation rate fell to normal, as did the blood picture. The latter change occurred much more rapidly.

In cases 7, 8 and 9 there was a new picture, one of massive destruction of tissue and necrosis of bone. After surgical intervention the rate of sedimentation increased markedly, to be followed by a more gradual return to normal. This secondary rise may be assumed to be the result of the increased damage to the tissues incident to any surgical procedure. The blood picture in these cases is that of any septic process. The last 2 cases (10 and 11) were representative of chronic mastoiditis in acute

exacerbation. In these, as is to be expected, only moderate changes in the rate of sedimentation were seen. The effect of surgical intervention on these is in all respects that described.

From the laboratory work presented one cannot draw any definite conclusions as regards any correlation between the blood picture and the sedimentation; however, the blood picture does seem to be more responsive to the acute inflammatory process, while the sedimentation rate appears to give a truer picture of the degree of destruction of tissue.

CONCLUSION

It may be tentatively observed that the rate of sedimentation will be in direct proportion to the amount of tissue destroyed. It is evident that should this statement be corroborated by further work there is presented here an aid to the diagnosis of progressive destructive disease of the middle ear and mastoid.

NASAL ACCESSORY SINUSES AS FOCI OF INFECTION IN ARTHRITIS

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In the literature considerable difference of opinion is expressed in regard to the importance of the accessory nasal sinuses as the primary or principal foci of infection in infectious arthritis. Snyder, Fineman, and Traeger,[1] in a study of 386 routine unselected patients with chronic arthritis, found that 93 (practically 1 of every 4) had sinusitis, proved roentgenologically and clinically. The disease of the sinuses was most frequently asymptomatic, or "silent." Of these 93 patients, 51 received rhinologic treatments of either a conservative or a radical nature. The arthritis in these 51 patients, with few exceptions, improved greatly as a result of rhinologic treatment of the diseased sinuses. In 262 of the 386 patients (68 per cent) roentgenologic examination revealed slight, moderate or extreme changes suggestive or indicative of sinal disease. Of these 262 patients, 126 were subjected to rhinologic examination, and 93 were found to have clinical evidence of active sinusitis. In reviewing the results in this series of patients, Hurd [2] found that 47 patients who had practically no treatment directed toward improving the condition of the sinuses showed little or no evidence of improvement in the arthritis. Of the 28 patients who had medical treatment directed toward eradicating the sinusitis, 71 per cent showed great or extreme improvement in their arthritic symptoms, and of the 18 patients who had surgical measures directed toward eradicating the sinusitis, 89 per cent showed great or extreme improvement in their arthritic symptoms. In a later paper Hurd [3] expressed the belief that patients who have infected

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1. Snyder, R. G.; Fineman, S., and Traeger, C.: Sinusitis in Chronic Arthritis, Laryngoscope **42**:682-694 (Sept.) 1932.

2. Hurd, L. M.: The Nasal Accessory Sinuses as Foci of Infection in Chronic Arthritis, Ann. Otol., Rhin. & Laryng. **42**:39-46 (March) 1933.

3. Hurd, L. M.: The Nasal Accessory Sinuses as Foci of Infection in Arthritis, M. Clin. North America **21**:1683-1687 (Nov.) 1937.

sinuses and rheumatoid arthritis frequently fail to show improvement until disease of the sinus has been removed, although every other condition that might have caused the arthritis has been either excluded as a possibility in differential diagnosis or corrected. Hurd found that the ethmoid sinuses were always the site of an infection and that most frequently there was disease in the maxillary sinuses and occasionally disease in the frontal or the sphenoid sinus in association with that in the ethmoid sinus. He stated further that "in this series, more than two thirds of the cases had previously had tonsillectomies without benefit to the arthritis," and he concluded that the tonsils were removed because they were easily accessible, whereas the sinuses were overlooked, to the detriment of the patient.

The aforementioned reports seem to be representative of the opinions of those who are most strongly convinced that sinusitis is an important etiologic factor in infectious arthritis.

Anderson,[4] on the other hand, is a representative of an opposite point of view. He reviewed 400 consecutive cases of suppurative disease of the paranasal sinuses and in 200 cases of sinusitis without other foci of infection found rheumatism coincident with infection of the upper part of the respiratory tract in only 3 cases and bronchitis in 1. Of the 200 cases in which other foci of infection were present complications occurred in 52 that might have been caused by focal infection. Anderson concluded from his study that in patients with infection of the teeth, tonsils or prostate gland diseases attributable to focal infection were of fairly frequent occurrence but that such diseases were almost entirely absent when sinusitis was the only known focus. He stated that from clinical observation he thought one can occasionally find a sinus acting as a focus of infection but that, since this is so rare an occurrence, one might, unless careful examination and conservative investigation reveal evidence of such a relation, consider it nonexistent. Anderson was greatly impressed with the number of instances in which an operation destructive of the physiologic function of the nose was done without subsequent benefit to the patient on the basis of roentgenologic evidence of suppurative disease of the paranasal sinuses.

Proponents of the hypothesis that suppurative disease of the paranasal sinuses is an important factor in the production of chronic arthritis object to conclusions such as Anderson has drawn. The reasons for their objections are, first, such patients present themselves with sinal disease as the chief complaint, and in the majority of patients with chronic arthritis sinusitis has been found to be of the "silent type,"

4. Anderson, C. M.: Suppuration in the Paranasal Sinuses as a Factor in Focal Infection: Review of Four Hundred Cases, J. A. M. A. **94**:1889-1891 (June 14) 1930.

producing no local signs or symptoms; second, in the groups studied, insufficient attention has been given to age, economic status, race, physique, previous and concomitant diseases and the variation in etiologic factors in different sections of the country.

In an attempt to discover what validity might be present in these objections, we resolved to study 100 consecutive cases of infectious arthritis. The patients included in this study had infectious (atrophic, rheumatoid) arthritis. Most of the patients had chronic infectious arthritis and showed evidence of inflammation of the synovial and periarticular tissues, roentgenographic evidence of atrophy of bone and narrowing of the joint space, limitation in the range of movement, with deformities, and ankylosis of some of the affected joints. A few of the patients had subacute inflammation of the synovial and periarticular tissues. Symptoms produced by aggravation of the infectious arthritis by trauma from too much activity were disregarded if the primary disease was caused by infectious arthritis.

It was thought that a group of 100 cases would be comparable to the group of Snyder, Fineman, Traeger and Hurd; although these authors stated that 386 patients were examined roentgenologically, it is obvious that not more than 126 of them received competent rhinologic examination.

In our group, anteroposterior and lateral roentgenograms of the sinuses were made in all cases with the additional use of stereoscopic plates if the roentgenologist thought it necessary for diagnostic purposes. A careful rhinologic history was taken, and the nose was inspected for the presence of secretion, inflammatory changes in the mucous membrane, edema or fibrosis of the submucosa or an appearance suggesting hypertrophic or hyperplastic changes in the superficial epithelium. The nasal chambers were then thoroughly cocainized, and the natural ostiums of the maxillary and sphenoid sinuses were tested for patency with a probe, which was then inspected for the presence of adherent secretions. The nasal chambers were thoroughly cleaned with a suction cannula, particular attention being paid to the choanal region. Gentle suction was then applied to the whole nasal chamber, such changes being made in the position of the head as tended to facilitate drainage from the various paranasal sinuses. The choanae were examined with a postrhinoscopic mirror to see if any secretion had been withdrawn from the posterior ethmoid-sphenoid group of sinuses. The upper pole of the nose, the middle meatus and the floor of the nose were then submitted to anterior rhinoscopic examination for the presence of secretion. If a suspicious secretion was noted in any of these examinations, this procedure was repeated on several successive days. If the discharge persisted, the antrums were punctured and lavaged and the sphenoid sinuses canalized and lavaged with 4 cc. of physiologic solution of sodium

chloride. If the results of rhinoscopic examinations were negative but evidence of a thickened membrane in the antrums was found on roentgenologic examination, these were lavaged. Investigation of the cytologic characteristics of the sinuses as suggested by Sewall [5] was not attempted; we have not found that attempts at especially refined methods of diagnosis have yielded worth while clinical results. If cysts in the antrum alone were reported by the roentgenologist, their presence was ignored, as Pattee [6] demonstrated by culture of the aspirated contents of such cysts that they are sterile.

DATA CONCERNING ONE HUNDRED CASES OF ARTHRITIS IN WHICH DETAILED RHINOLOGIC EXAMINATION WAS PERFORMED

The group considered was made up of patients from various parts of the country: 22 from Minnesota; 14 from Wisconsin; 9 from Illinois; 8 each from Iowa, North Dakota and Indiana; 5 each from Montana and Michigan; 2 each from Colorado, Oklahoma, California and Kansas, and 1 each from South Dakota, Pennsylvania, New York, West Virginia, Arizona, New Mexico, Alabama, Nebraska, Arkansas, Texas and Wyoming and from Alberta and Manitoba, Canada. Although the majority of patients were from the Central states, there was a sufficiently wide representation so that any local factor probably could be disregarded. There were 42 cases in which roentgenologic examination indicated changes in the paranasal sinuses and in which the condition might have been caused by infection. Of the 42 patients, 17 had related a history suggesting suppurative disease in the paranasal sinuses, and 4, hay fever or vasomotor rhinitis. In other words, 21 patients in the group of 42 presented roentgenologic evidence and a history of symptoms of rhinitis.

In 20 cases in the whole group of 100 clinical investigation yielded definite evidence of suppurative disease of the paranasal sinuses. Of these 20 cases, 19 were in the group of 42 in which positive roentgenologic evidence was obtained; in 1 case in which suppurative involvement of the sphenoid and the posterior ethmoid sinuses was found, the results of roentgenologic examination of the paranasal sinuses were negative. In 17 cases in which roentgenologic evidence of sinusitis was obtained, clinical investigation did not yield evidence of sinusitis. In 6 cases in which roentgenologic evidence of sinusitis was obtained, the examination

5. Sewall, E. C.: Cytological Examination in the Diagnosis of Infection in the Nasal Accessory Sinuses, Ann. Otol., Rhin. & Laryng. **37**:642-660 (June) 1928.

6. Pattee, G. L.: The Relative Value of Various Clinical and Laboratory Methods in the Diagnosis of Chronic Maxillary Sinusitis, Thesis, Universitiy of Minnesota, 1933.

was reported incomplete. In 3 cases in the group of 20 in which clinical investigation had yielded evidence of sinusitis, removal of infected teeth or local treatment of the nose apparently eradicated suppurative disease in the paranasal sinuses, but in none of these cases was a favorable effect on the course of the arthritis noted.

In the group of 20 cases there were 2 in which evidence of sinusitis was obtained on clinical investigation and in which, in our opinion, the presence of cardiac complications contraindicated operative intervention. In 15 cases operation directed toward eradicating infection in the paranasal sinuses was advised. Of the 15 patients, 12 were operated on at the Mayo Clinic; 3 refused surgical therapy. Of these 12 patients, 9 had related a history suggestive of involvement of the paranasal sinuses by suppurative disease, 3 having the so-called silent type of sinusitis. In 5 cases in which an operation was performed on the sinus, a tonsillectomy was performed within a week. Four of the patients reported almost complete relief from arthritic symptoms within two weeks after the operation on the paranasal sinuses, and after two years they reported that arthritic symptoms had almost ceased. In 7 cases in which an operation on the sinus alone was performed, there were apparently no other foci of infection. Two of the patients experienced almost complete relief from arthritic symptoms within two weeks after operation on the paranasal sinuses and were still in good health after two years. A third patient reported almost complete relief from arthritic symptoms within two weeks after operation, but after six months the symptoms began to return, and at the end of two years the arthritic symptoms were as severe as, if not worse than, they were before the sinusostomy was performed. It is of interest to note that if improvement did not appear within the first two weeks after surgical removal of a focus of infection, none appeared subsequently.

SURGICAL PROCEDURES PERFORMED ON THE TWELVE PATIENTS

In the first case, anterior ethmoidectomy (intranasal) was performed on the left; submucous resection and an intranasal operation on all sinuses was performed in the second case. The pathologic report on tissue removed from the sinuses was: inflammatory adenomyxomatous polyps, with few eosinophils. Bilateral antral windows were made in the third case. The pathologic report on the lining membrane removed at operation was: inflammatory adenomatous tissue. In the fourth case an antral window was made on the right. The pathologic report was: inflammatory tissue. The patient in the fifth case was subjected to right sphenoidectomy and posterior ethmoidectomy. The pathologic report was: inflammatory tissue. In the sixth case, bilateral antral windows were made, and anterior and posterior ethmoidectomy and bilateral

sphenoidostomy were performed. The pathologic report was: inflammatory tissue. Bilateral antral windows were made in the seventh case and an antral window on the left in the eighth case. In the ninth case anterior and posterior ethmoidectomy and bilateral sphenoid sinusostomy were performed. In the tenth case, bilateral antral windows were made, and anterior and posterior ethmoidectomy and bilateral sphenoid sinusostomy were performed. Surgical measures in the eleventh case consisted of anterior ethmoidectomy and incision of the frontal and the sphenoid sinus. The pathologic report was: inflammatory tissue with numerous plasma cells in the infiltrate. An antral window was made on the right in the twelfth case.

COMMENT

Although in 42 of 100 cases of infectious arthritis roentgenologic examination revealed cloudiness graded 1 to 4 (on a basis of 1 to 4) in one or more of the paranasal sinuses, in only 20 cases did clinical investigation give evidence of suppurative disease in the paranasal sinuses, and in 1 of these cases roentgenologic examination gave negative results. It would be interesting to know what the incidence of positive results would be on roentgenologic examination of sinuses in an unselected group of the general population. It is possible that in 42 per cent of cases suggestive changes would be found.

Of 12 patients with infectious arthritis who were subjected to operation on the paranasal sinuses in an attempt to eradicate a focus of infection, 6 experienced permanent improvement that might be attributed to the operation, but, of these 6, 4 had tonsillectomy also performed at nearly the same time. One other patient experienced transient improvement in arthritic symptoms after operation on the sinuses.

SUMMARY

Although in an unselected group of 100 cases of infectious arthritis 20 cases of clinically proved sinus disease were found, the results experienced by the 12 patients who were operated on do not support the conclusion that sinusitis is the principal focus of infection in cases of infectious arthritis. If disease of the sinuses is found, an attempt should be made to eradicate it, but too optimistic an outlook as to the results of this type of therapy should be avoided. It would seem to be definitely an error, however, to assume that the sinuses can be ignored as possible foci of infection in cases of infectious arthritis.

ABSCESS OF THE BRAIN FOLLOWING TONSILLITIS AND RETROPHARYNGEAL ABSCESS

REPORT OF A CASE AND REVIEW OF THE LITERATURE

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Intracranial complications of infections of the tonsil and of the peritonsillar and the pharyngeal region are not common. Infections terminating in meningitis, thrombosis of the cavernous sinus or thrombosis of the internal jugular vein with extension into the cranial sinuses are comparatively more frequent than those terminating in abscess of the brain. A review of the literature discloses only 9 instances of the latter since 1921.

Kernan [1] reported the case of a patient who was sick seventeen days. Autopsy revealed abscess of the tonsillar region on the left side and thrombosis of the internal jugular vein extending into the lateral sinus beyond the knee. Both the petrosal and the cavernous sinuses contained partly broken-down clots. An abscess of the left temporal lobe was observed.

Housden [2] in 1929 described a case in which, after acute tonsillitis, the patient seemed to get well and was out of bed for three weeks. However, nine weeks after the onset the patient suddenly died. Autopsy revealed an abscess in the left temporosphenoid lobe. Housden stated that there is little or no reference in the literature to this complication.

Goodyear [3] reported the case of an 18 year old youth who three weeks before examination had had a peritonsillar abscess incised. On his admission to the hospital, the right side of the neck was incised, and pus was found, with thrombosis of the external jugular vein. Thirteen days after admission right hemiplegia developed. The patient became unconscious. The left frontal lobe was explored and no pus found. The right hemisphere of the cerebellum was covered with pus. The patient died fifteen days after admission. Autopsy revealed an abscess 3 cm.

From the otolaryngologic service of Dr. George D. Wolf, Sydenham Hospital.

1. Kernan, J. D., Jr.: Cavernous Sinus Thrombosis, Secondary to Peritonsillar Abscess, Laryngoscope **31**:259, 1921.

2. Housden, E. G.: Cerebral Abscess Following Acute Tonsillitis, Brit. M. J. **1**:1158 (June 29) 1929.

3. Goodyear, H. M.: Peritonsillar Abscess, Thrombosis of External Jugular Vein, and Brain Abscess, Laryngoscope **39**:674 (Oct.) 1929.

in diameter and 1 cm. deep immediately over the pituitary body. No evidence of thrombosis in the cavernous or other venous sinuses was seen. The right internal jugular vein was not involved. General suppurative meningitis was evident.

Two cases are reported in which abscess of the brain followed tonsillectomy: Johnson [4] reported a case of cerebral abscess in a boy of 7 years on whom a tonsillectomy was performed five and one-half weeks after the onset of acute tonsillitis. The first five postoperative days were uneventful; then a chill and sudden rise in temperature occurred. On the tenth day complete left-sided hemiplegia was present. Culture of the spinal fluid was sterile. Autopsy revealed an abscess of the right hemisphere.

Cadbury and Siddall [5] reported a case in which cerebral abscess followed tonsillectomy performed with the patient under local anesthesia. Convalescence was normal for five days, at the end of which sudden epileptiform convulsions occurred. At autopsy, abscesses were seen in the right frontal, the right parietal and the right temporal lobe.

Turner and Reynolds,[6] among 55 cases of intracranial infection secondary to foci in the head or neck, observed only 1 in which the original focus was the tonsillar region. In their "Case VIII" a woman aged 34 seven days after an attack of sore throat had spontaneous rupture of an abscess of the left tonsil. Fever persisted, and edema of the left upper and lower eyelids and proptosis of the left eye occurred on the fourteenth day. She died on the sixteenth day of her illness. Autopsy revealed purulent basilar meningitis extending from the hypophysis under the tentorium cerebelli to the posterior cistern. Both cavernous sinuses contained pus. An extradural abscess was noted on the left. The middle ears were normal. A small abscess appeared immediately above the upper pole of the left tonsil. The cervical glands were enlarged. The internal jugular vein was not thrombosed. Pyemic abscesses were present in both lungs.

In addition, Turner and Reynolds cited Wessely,[7] who collected 8 cases of intracranial complications of tonsillar infection from the literature and added 2 of his own. The 10 cases comprised 1 of abscess of the temporal lobe, 4 of septic thrombosis of the cavernous sinus and 5 of purulent leptomeningitis.

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4. Johnson, W. C.: Cerebral Abscess Following Tonsillar Infection, Brit. M. J. **2**:13 (July 5) 1930.

5. Cadbury, W. W., and Siddall, A. C.: Cerebral Abscess Following Tonsillectomy, China M. J. **44**:910 (Sept.) 1930.

6. Turner, A. L., and Reynolds, F. E.: Intracranial Pyogenic Diseases: A Pathological and Clinical Study of the Pathways of Infection from the Face, the Nasal and Paranasal Air-Cavities, Edinburgh, Oliver & Boyd, 1931.

7. Wessely, E.: Die endocranielle Komplikation nach Peritonsillitis, Ztschr. f. Hals-, Nasen- u. Ohrenh. **9**:439, 1924.

Grabscheid [8] in 1934 reported a case of peritonsillar abscess (on the left side) which resulted in an abscess of the frontal lobe and death three months after the onset of the original sore throat.

B. Kecht [9] in a report of 7 cases of endocranial involvement following infection of the mouth and pharynx cited only 1 in which abscess of the brain occurred. There were 2 cases of dental, 1 of pharyngeal and 4 of tonsillar origin.

REPORT OF CASE

A. W., a white man aged 28, was first seen by me on Feb. 28, 1938. He gave a history of sore throat since February 21. His initial temperature of 103 F. had subsided in a few days, and on the morning of February 28 it was 99.2 F., rectal. He swabbed his throat with a 10 per cent solution of mild protein silver, and shortly thereafter he had a chill lasting fifteen minutes and his temperature went up to 103 F. Examination revealed an apprehensive patient complaining of slight pain in the left side of his throat which became worse on swallowing. The ears were normal. The mucous membrane of the nose was only slightly congested. The tonsils were moderate sized and acutely inflamed; no edema of the throat was seen. Pressure on the anterior tonsillar pillars failed to express pus. A hot saline gargle and large doses of sulfanilamide were prescribed. From Febraury 28, to March 5, the temperature fluctuated between 102 and 103.6 F. The administration of sulfanilamide was discontinued on March 3 because of cyanosis. On March 5 tenderness developed over the middle of the left sternocleidomastoid muscle. The patient was advised to enter the hospital and was admitted on March 5 to Sydenham Hospital.

Physical examination on admission revealed no abnormality except for the condition in the neck, throat and nose. The oropharynx was moderately injected and the tonsils hypertrophied and red. The left tonsil protruded slightly more into the pharynx than the right tonsil. The mucous membrane of the nose was markedly injected and edematous; the septum was deviated to the right; no free pus was found in either chamber. Slight tenderness was elicited in the left anterior cervical region, but no mass was felt. The reflexes were slightly depressed. No nuchal rigidity or other neurologic sign was found. The temperature on admission was 104.4 F., the pulse rate 90 and the respiratory rate 24. A blood count showed 4,420,000 red cells, with 87 per cent hemoglobin, and 27,550 white cells, with 3 per cent band forms, 85 per cent segmented cells, 3 per cent lymphocytes and 9 per cent monocytes. Urinary findings were as follows: specific gravity, 1.023; acid reaction; 1 plus reaction for albumin; negative reaction for sugar, acetone and diacetic acid, and 1 to 3 red and 4 to 6 white cells per high power field. A smear of material from the left tonsil showed some pus cells and numerous diplococci, and culture of material from the left tonsil showed a nonhemolytic streptococcus. The patient was given 15 grains (0.97 Gm.) of sulfanilamide every four hours, six doses every day, and hot saline irrigations every two hours.

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8. Grabscheid, E.: Ein Beitrag zur endokraniellen Komplikation nach Rachenphlegmone, Klin. Wchnschr. **13**:1017 (July 14) 1934.

9. Kecht, B.: Zur Kenntnis und Behandlung der zum Endokranium aufsteigenden Eiterungen der Mundhöhle und des Rachens, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **141**:275, 1936.

The temperature remained at 104 F. until the third day in the hospital, when it went up to 105 F. and later dropped to 103. The only complaint was a sense of fulness in the left side of the throat. An examination on March 8 was reported as follows: "The swelling behind the left tonsil is fuller and appears to be dissecting in the fascial plane to the midline. There is a sensation of fluctuation at a point above the level of the base of the tongue." Medical examination gave essentially negative results. A blood culture was reported negative. A blood count revealed 32,600 white cells, with 16 per cent band forms, 72 per cent segmented cells, 11 per cent lymphocytes and 1 per cent monocytes. Ten cubic centimeters of prontosil[10] was given intramuscularly three times a day. On March 8, fifteen days after the onset of the sore throat, with the patient under local anesthesia, an incision was made into the swelling behind the left tonsil, and about 1½ ounces (45 cc.) of thick, creamy pus was obtained, from which a nonhemolytic streptococcus was cultured. The temperature that evening was 106 F., and a blood culture was taken. This was reported negative after seventy-two hours. Roentgenograms of the chest and mediastinum likewise showed no abnormality. The next day the patient was given a transfusion of 300 cc. of whole blood; the incised wound in his pharynx was reopened, and about ½ ounce (15 cc.) of seropurulent material was expressed. Examination of the fundi showed only slight dilatation of the retinal veins. Medical examination gave negative results except for the local findings. The temperature rose that afternoon to 106.4 F. Throughout this period the patient was euphoric, felt well and complained of nothing but a sense of fulness in the left side of the neck.

The highest temperature on March 11 was 103.2 F. In view of the definite improvement in the appearance of the pharynx and the continued elevation of temperature, sense of fulness and tenderness over the left sternocleidomastoid muscle, an exploration of the neck was advised. The region of the left carotid sheath was exposed on March 12, with the area under local anesthesia. No thrombus was felt, and the vein was easily compressible. A localized collection of pus was not found in any fascial plane. A drain was inserted into the lower angle of the wound and the patient returned to bed.

From March 12 to 19 the temperature varied between 102 and 103 F.; the pulse rate varied between 80 and 100, and the respiratory rate was 24. The patient felt well, ate well and had no complaints. On March 20 the temperature started to climb again, reaching 104 F.; the pulse rate was between 90 and 120. The urine gave a 1 plus reaction for albumin and contained a few red cells and 10 to 20 white cells per high power field. The blood pressure was 110 systolic and 56 diastolic. On March 21, the temperature was above 104 F. Verbal aphasia developed, but the patient responded to commands. On March 22, the aphasia became more pronounced, and slight rigidity of the neck was present. On March 23, the condition was worse. The temperature ranged between 104 and 105 F. and the pulse rate from 110 to 120.

The observations made at an ophthalmologic consultation on March 23 were reported as follows: "The right optic disk is slightly swollen, with blurred margins, and reddish gray. The veins are dilated but not tortuous. No hemorrhages or exudates are seen. The left optic disk is less blurred than the right; the left fundus appears the same as the right."

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10. Disodium 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′,-6′-disulfonate. This is the substance previously known as prontosil soluble and now to be called neoprontosil.

The results of a neurologic examination on the same day were reported by Dr. Israel Wechsler as follows: "The patient is dull and does not respond to questions. He responds to commands by gesture but not to verbal commands. He has verbal, sensory and nominal aphasia. Nuchal rigidity and a bilateral Kernig sign, more marked on the right side, are present. There is a suggestion of weakness of the right facial nerve. The deep reflexes are present. The abdominal reflexes are present, but those on the right are less active; Babinski's sign is not present. The response to a pinprick is present but is less marked on the right side. No obvious motor palsy is found. There is a suggestion of right hemianopia, as the patient does not blink his eyes as fingers come from the right side. The margins of the optic disks appear blurred. The findings justify the diagnosis of meningitis and suggest the presence of a focus of involvement in the supramarginal gyrus on the left side."

A neurosurgical consultation by Dr. Ira Cohen led to the following report: "While an abscess of the left frontal lobe of the brain is a possibility, I favor the diagnosis of localized subarachnoid infection more. It is impossible to distinguish between a metastatic lesion and one due to retrograde sinus thrombosis. If the latter condition exists, the damage is already done, and I feel that a sinal operation is contraindicated. There is no present indication for surgical intervention."

The initial spinal tap, performed on March 23, gave the following manometric readings:

	Mm. of Water
Initial pressure	380
Right jugular pressure	520
Right jugular pressure on release	340
Left jugular pressure	500
Left jugular pressure on release	320
Bilateral jugular pressure	420
Pressure on release	320
Final pressure	160

The fluid was slightly turbid. Examination showed albumin (3 plus), a trace of globulin, 63 mg. of sugar and 300 cells, mostly polymorphonuclears. The smear and culture were negative. At no time was a positive growth obtained on culture of the spinal fluid or any organism seen on direct smear. On March 24 the cell count was 600, and on March 25, 750.

Other taps produced xanthochromic fluid or frank blood.

A roentgenogram of the head taken on March 23 was reported on as follows: "The sinuses are clear; the skull shows an increase in convolutional markings."

From March 23 to March 29 the patient continued in a coma. He lost control of his sphincters on March 24. During this period repeated spinal taps were done. The temperature ranged between 104 and 105 F.; the pulse rate was consistently high. Sulfanilamide was given by mouth and prontosil [10] intramuscularly.

The patient died on March 29, thirty-six days after the onset of his illness and eight days after the onset of the neurologic signs.

A summary of the report made at autopsy follows: Permission was obtained for autopsy on the head and neck only.

Gross Examination.—Neck: The left carotid sheath was identified and found intact. The carotid artery lay laterally, and the internal jugular vein lay medially. On incision of both structures liquid blood escaped from them. Thick creamy pus oozed from all fascial planes.

Head: The dura was not adherent to the calvarium; the superior sagittal sinus contained liquid blood. On removing the dura, the right cerebral hemisphere was seen to have somewhat flattened gyri and narrowed sulci. The pial vessels were extremely engorged, but no exudate was noticed. On removal of the dura from the left side a large amount of creamy grayish pink pus escaped from the subdural space. This was seen to come from an area reaching from the postcentral gyrus to the frontal lobe and thus including the entire frontal and most of the parietal and temporal lobes. The pus was present also on the medial aspect of the left cerebral hemisphere. The occipital lobe was entirely free except for some engorgement of the pial vessels. The diseased area was converted into a mass of creamy pus, which destroyed the normal architecture, eating into the gray matter. The base of the brain was free except for small areas around the sylvian fissure. The interpeduncular sinus and the pons and medulla were entirely free from pus, showing only injection of the vessels. The brain was removed and the inferior sagittal sinus found to contain liquid blood. The right transverse and sigmoid sinuses contained liquid blood. The left transverse and sigmoid sinuses to the torcular Herophili were empty except for creamy gray-yellow pus. The inferior petrosal sinuses on both sides and the superior petrosal sinus on the right side were free. The left superior petrosal sinus contained pus, as did the cavernous sinus, especially on the left side. The hypophysis was converted into a purulent mass. The dura was removed, and no trace of fracture was noted. Several deep vascular channels were present in the bones. Both middle and internal ears were entirely free. The sphenoid sinus was opened through the basal part of the sphenoid bone, and the mucous membrane sprang into the osseous opening. Pus under pressure escaped on incision of this sac.

Brain: The brain weighed 1,250 Gm. and was sectioned according to the Virchow method. No gross pathologic changes were noted except two 0.5 cm. abscesses located subcortically near the motor region and two smaller abscesses in the vermis of the cerebellum. The external surface of the cerebellum was entirely free except for a small amount of exudate on the superior surface of the vermis. The basilar vessels were entirely free, as was the circle of Willis. The ventricles showed no gross pathologic changes except slight injection of the choroid plexus; they contained clear liquor.

Bacteriologic Examination.—Cultures and a smear of material taken from the meninges showed no bacteria, but a large number of polymorphonclear cells were present.

Microscopic Examination.—Sphenoid Sinus: Sections from the sphenoid sinus showed partly degenerated columnal epithelium supported by fibrillar connective tissue. Leukocytes and plasma cells were scattered throughout the fibrous tissue. In some places the epithelium formed a single row; in others, several rows with smaller cells at the base.

Sigmoid Sinus: Sections failed to reveal any epithelial covering. The surface was covered with young granulation tissue which surmounted fibrillar and moderately cellular connective tissue.

Cerebrum: The arachnoid was covered with leukocytes and fibrin. The adjoining cortex was the seat of extravasation, necrosis and leukocytic infiltration. Some vessels were filled with leukocytes; others showed endothelial hyperplasia;

still others were thrombosed or undergoing necrotic changes. Scattered throughout the section were small and large abscesses with neutrophils and scavenger cells.

Cerebellum: Sections of the cerebellum presented a picture similar to that of the cortex.

Diagnosis.—The following conditions were noted: (1) status after incision of the right anterior triangle of the neck, with infection of the fascial plane; (2) localized purulent meningoencephalitis of the left temporoparietofrontal lobe of the cerebrum; (3) cortical abscesses in the cerebellum; (4) empyema of the left transverse, sigmoid, cavernous and superior petrosal sinuses, and (5) the empyema of the sphenoid sinus.

ROUTES OF INFECTION

B. Kecht [9] stated that the condition may be transmitted, first, by way of loose tissue of the parapharyngeal space, as extension abscesses, or, second, by venous passages, (*a*) the pterygoid plexus and the connecting venous channels or (*b*) the internal jugular vein or sigmoid sinus.

The most probable route of extension in the case presented was through the pharyngeal plexus into the pterygoid and thence into the cavernous sinus by way of the vesalian vein or the rete foraminis ovalis. From the cavernous sinus the infection spread through the left superior petrosal sinus into the transverse sinus, extending backward to the torcular Herophili and forward to the sigmoid portion of the lateral sinus. The abscesses of brain tissue resulted from extension along the veins draining the involved areas. The normal roentgenogram taken at the onset of aphasia, the extent of intracranial destruction and the intact state of the roof of the sphenoid sinus led me to believe that the empyema of the sphenoid sinus was not the cause of the involvement of the brain.

COMMENT

Localized thrombosis, abscess or general sepsis follows acute tonsillitis with sufficient frequency to be given first consideration when resolution of an acute tonsillar infection does not take place normally. When an abscess is formed and its cavity is entered and pus found and evacuated, prompt subsidence of the infection is expected. In the case reported there was no improvement. Several days of septic temperature followed the incision of the abscess. The possibility of venous thrombosis or dissecting abscesses of the neck was considered. The exploration of the left side of the neck and the left internal jugular vein failed to reveal any extension of the infection. Blood cultures taken with the temperature at various levels were persistently negative. Although the possibility of meningitis was considered, it was not until the twenty-second day of the illness, when aphasia developed, that abscess of the brain was considered.

PNEUMOTHORAX AND MEDIASTINAL EMPHYSEMA COMPLICATING TRACHEOTOMY

MERRILL W. MICHELS, M.D.
DETROIT

Pneumothorax and mediastinal emphysema following tracheotomy are apparently more common than the literature would suggest. The fact that only a few cases are reported signifies not that an accident of this type is rare but rather that it is seldom recognized. The failure to recognize pneumothorax and mediastinal emphysema often results in unnecessary death.

Reviews of large series of tracheotomies fail, for the most part, to mention pneumothorax or mediastinal emphysema. Figi,[1] in reporting a series of 200 consecutive tracheotomies performed at the Mayo Clinic, made no mention of either of these conditions. Pneumothorax complicating tracheotomy has been reported by Iglauer,[2] Myerson,[3] Wiethe[4] and Simpson.[5] Leiner[6] cited 9 cases of mediastinal emphysema following tracheotomy in children. After bronchoscopic procedure, pneumothorax has been reported to occur as a more frequent complication from direct perforation of the pleura.

Several anatomic factors regarding the mechanisms responsible for pneumothorax must be considered. In the child, the possibilities for injury in performance of tracheotomy are greater than in the adult. The trachea is smaller and more deeply placed and is movable, with a length of 4 to 6 cm. as compared with the length in the adult of 10 to 12 cm. The right pleural dome reaches higher in the neck than the left. Cervical

From the Department of Otolaryngology, Harper Hospital.

1. Figi, F. A.: Tracheotomy: A Study of Two Hundred Cases, Ann. Otol., Rhin. & Laryng. **43**:178 (March) 1934.

2. Iglauer, S.: Accidental Pneumothorax During Tracheotomy, Ann. Otol., Rhin. & Laryng. **24**:303, 1915.

3. Myerson, M. C.: Perforation of the Bronchus with Notes on Bronchoscopic Anatomy, Ann. Otol., Rhin. & Laryng. **41**:1203 (Dec.) 1932.

4. Wiethe, C.: Ueber Pneumothorax bei Tracheotomia inferior, Monatschr. f. Ohrenh. **67**:57 (Jan.) 1933.

5. Simpson, W. L.: Pneumothorax Complicating Tracheotomy in Fulminating Laryngotracheobronchitis, Arch. Otolaryng. **26**:411 (Oct.) 1937.

6. Leiner, C.: Mediastinales Emphysem bei tracheotomierten Kindern, Jahrb. f. Kinderh. **58**:448, 1903.

herniation of the lung, according to Brenner,[7] commonly results from a weakening of Sibson's fascia between the anterior scalenus and the sternocleidomastoideus muscle, making this region especially liable to injury.

Wright [8] demonstrated that the thorax is functionally one cavity and that pressures in the two pleural cavities are always the same, within practical limits. If pneumothorax occurs, air will enter the thorax by the glottis and the new opening as the chest enlarges during inspiration. Little air enters the lungs, and asphyxia rapidly supervenes if the pneumothorax opening is large enough. The smaller the pneumothorax opening and the greater the compensatory increase in breathing, the more likely is air to enter the lungs. Even though the opening is small, the patient may succumb if he cannot respire deeply enough to make his vital capacity equal his tidal air.

In 1919, Berkley and Coffen [9] observed extensive subcutaneous and interstitial emphysema during an epidemic of influenza and bronchopneumonia. Their autopsies showed that air gained access to the mediastinum from the lung without passing through the pleura. Rupture of a distended air sac and a dissection of the air from the sac back to the hilus occurred. Streaks of air were seen leading off from the sacs, which followed the course of the large vessels. Kelman [10] determined the pathway of emphysema in rabbits by doing tracheotomy and varying the intratracheal pressure.

Actual pneumothorax was produced in dogs by Joannides and Tsoulos,[11] who varied the intrapulmonic pressure. With increased pressure, acute tension pneumothorax was produced. The authors observed large air-containing blebs near the hilus of the lungs, at the junction of the parietal and the visceral pleura. On rupture of these blebs, pneumothorax was produced. A high percentage of air embolism was found in animals subjected to increased intrapulmonic pressure.

More recently, Macklin [12] produced pneumothorax in cats by local overinflation of the pulmonary substance. The living lung was fixed

7. Brenner, E. C.: Pediatric Surgery, Philadelphia, Lea & Febiger, 1938.

8. Wright, S.: Applied Physiology, ed. 5, New York, Oxford University Press, 1934.

9. Berkley, H. K., and Coffen, T. H.: Generalized Interstitial Emphysema and Spontaneous Pneumothorax, J. A. M. A. **72**:535 (Feb. 22) 1919.

10. Kelman, S. R.: Experimental Emphysema, Arch. Int. Med. **24**:332 (Sept.) 1919.

11. Joannides, M., and Tsoulos, G. D.: Etiology of Interstitial and Mediastinal Emphysema, Arch. Surg. **21**:333 (Aug.) 1930.

12. Macklin, C. C.: Pneumothorax with Massive Collapse from Experimental Local Over-Inflation of the Lung Substance, Canad. M. A. J. **36**:414 (April) 1937.

by insufflation with osmic acid vapor. His findings confirmed the work of previous investigators, demonstrating that air does not pass directly from the lung through the pleura to produce pneumothorax. He showed that air may pass from the mediastinum upward to the neck or downward to the retroperitoneal tissues and manifest itself as subcutaneous emphysema.

Boyd [13] revealed that the lymphatics may play a role in the dissemination of air. He observed air escaping into the interstitial tissues of the lung and being collected in the lymphatics in the form of tiny beads, which passed to the mediastinum.

Another explanation has been presented, by Schomer and Ehrlich,[14] who related that during normal respiration the gliding of the visceral over the parietal pleura causes a continuous shedding and regeneration of the pleural mesothelium. A slight defect in regeneration, accompanied by increased respiratory effort, may cause a tear in the visceral pleura and thus produce pneumothorax.

The possibility of direct injury to the pleural dome in performance of low tracheotomy is not too remote, especially if there has been any deviation from the midline. When expiration is impeded the pleural domes rise above the clavicles, and if the head is retroflexed they rise still higher. Wiethe [4] reported 2 cases of pneumothorax in a series of 50 inferior tracheotomies. By his studies on the cadavers of children, he determined that the pleura under normal relations can be injured only with difficulty during tracheotomy. However, roentgen studies made in cases of expiratory interference showed that the pleural apexes rose considerably above the margins of the clavicles. He duplicated the conditions of increased intrapulmonic pressure and revealed by injecting solution of formaldehyde into the trachea under pressure that the apex of the pleura can easily be injured in performing low tracheotomy. The observation has been made on several occasions that the pleura, unaccompanied by lung, actually protruded into the tracheotomy wound prior to incision of the trachea. This has been encountered by Iglauer,[2] by Neffson and Bullowa [15] and by me on 2 occasions (cases 4 and 5).

The opening of the cervical fascia in tracheotomy provides an opportunity for air to gain entrance into the mediastinum. As long ago

13. Boyd, W.: A Text-Book of Pathology, ed. 3, Philadelphia, Lea & Febiger, 1938.

14. Schomer, A., and Ehrlich, D. E.: Spontaneous Pneumothorax, M. Bull. Vet. Admin. **11**:206 (Jan.) 1935.

15. Neffson, A. H., and Bullowa, J. G. M.: Influenza with Simultaneous Bilateral Spontaneous Pneumothorax and Subcutaneous Emphysema, Arch. Otolaryng. **28**:388 (Sept.) 1938.

as 1884, Champney [16] performed a series of experiments on stillborn children to determine whether tracheotomy causes pneumothorax. He found that if the entry of air is impaired during forcible inspiration and at the same time the deep cervical fascia is opened a potential passage into the thorax becomes established. If while doing tracheotomy one makes a small incision in the pretracheal fascia and lifts the fascia away from the trachea, air is drawn into the mediastinum. If the inspiration is violent, air can pass through the mediastinum directly to the pleural sac.

Jackson [17] mentioned that stitching up the tracheotomy wound can cause mediastinal emphysema. The error is avoided by packing the wound widely open.

Thus it has been seen that pneumothorax and mediastinal emphysema may be established by several pathways. To bring them about by the intrapleural route, with direct rupture of the parenchyma and the pleura of the lung, requires a pressure of from 100 to 300 mm. of water, according to Kirshner.[18] The extrapleural route, with dissection of minute globules of air from the interstitial tissues along the vessels to the hilus, probably is a more common cause of their occurrence with increased intrapulmonic pressure. The dissection of air by way of the pretracheal fascia to the mediastinum, aided by the negative inspiratory pressure, affords another avenue of entry. The possibility of direct injury to the pleural dome during tracheotomy cannot be ignored.

After tracheotomy or any endoscopic procedure, the patient should receive careful attention. If increasing respiratory embarrassment becomes evident, an accurate evaluation of the physical findings is essential. Fluoroscopic or roentgen examination of the chest is indicated. The presence of a tracheotomy tube often obscures the clinical signs of pneumothorax. Pneumothorax may be recognized usually by the greatly diminished excursion on the affected side, by the displacement of the heart to the opposite side and by the diminution or absence of breath sounds. Percussion reveals a hyperresonant, tympanitic note; however, bronchi plugged with mucus may give rise to dulness. Should the cavity contain any fluid, a succussion splash is heard.

Mediastinal emphysema may be recognized by the crepitant sounds following each heart beat. Leiner [6] has noted the fine vesicular crepitation synchronous with the cardiac action in 9 cases of mediastinal emphysema in tracheotomized children. The area of cardiac dulness may be

16. Champney, F. H., cited by Iglauer.[2]

17. Jackson, C., and Jackson, C. L.: The Larynx and Its Diseases, Philadelphia, W. B. Saunders Company, 1937.

18. Kirshner, J. J.: Spontaneous Pneumothorax, Am. J. M. Sc. **196**:704 (Nov.) 1938.

replaced by an abnormally loud, sonorous, tympanitic percussion sound. The apex beat is often lost to inspection and palpation.

The treatment of tension pneumothorax is simple, but when unrecognized the condition often causes death. A small pneumothorax may cause no embarrassment and may become absorbed in a relatively short period. Tension pneumothorax, with a valvelike action permitting air to enter the pleural cavity but preventing its escape, requires some form of decompression. Periodic aspiration of the pneumothorax is advocated by some writers. The dangers of injury to the lung, of air embolism and of pleural shock are enhanced, however, by any unnecessary manipulation of the pleura. Aside from this objection, rapid decompression, which occurs with repeated pleural paracenteses, allows too great changes in intrathoracic pressure. As the intrathoracic pressure increases, the flow of venous blood in the thorax is retarded, and virtual mediastinal tamponade may ensue. Anoxia, air embolism and pleural shock are among the immediate complications of pneumothorax. If too rapid decompression is permitted, mediastinal flutter may take place.

Treatment by closed drainage obviates many of the objectionable features of periodic aspiration. Brenner [7] suggested a method of effecting closed drainage by inserting a dull silver needle (a blunt cannula or rubber catheter also may be used) just beyond the parietal pleura and connecting it with rubber tubing. The distal end of the tubing is placed in an antiseptic solution about 18 inches (46 cm.) below the level of the chest. Drainage is continued as long as air escapes. When the fluid rises in the tube and remains there, showing free fluctuations, a period without drainage may be tried by clamping the tube. If no respiratory distress follows, the needle may be withdrawn.

Concentrated dextrose on oil of cajuput has been introduced into the pleural cavity in cases of persistent pneumothorax when infection is absent.

The treatment of mediastinal emphysema is more complex and demands a special approach. Thorek [19] proposed an incision in the anterior aspect of the infrathyroid region, going down to the trachea. A suction apparatus is then improvised to withdraw the escaped air gradually from the mediastinum. Continuous suction may be used according to the circumstances.

Many of the complications incidental to tracheotomy could probably be eliminated by the preliminary introduction of a bronchoscope. This procedure converts a struggling, restless child into a quiet one with an adequate airway. The increased intrathoracic pressure incidental to obstruction is thus relieved, and sufficient time is allowed for an unhurried, careful tracheotomy.

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19. Thorek, M.: Modern Surgical Technic, Philadelphia, J. B. Lippincott Company, 1938, vol. 2.

REPORT OF CASES

During the past two years the following cases of pneumothorax and mediastinal emphysema complicating tracheotomy have been observed:

CASE 1.—W. Z., a 3 year old white boy, was admitted to Children's Hospital of Michigan on March 20, 1937, with cyanosis and shortness of breath. He had been well until three weeks before admission, when he suffered from a "sore throat." He was placed in bed and improved with symptomatic treatment. About a week later he had a temperature of 103 F., general malaise, increased irritability and a severe nonproductive cough. On the day of admission he became extremely dyspneic and cyanotic.

Examination showed an acutely ill boy, with signs of respiratory embarrassment and obstruction. There was a diminution of excursion of the left side of the chest, which was dull to percussion. Respiratory sounds were diminished in this region. Subcutaneous emphysema, over the entire left side of the chest, extended well into the axilla. Cardiac displacement was not present.

Roentgenograms of the chest showed a small screw in the left main bronchus, with secondary emphysema, and some atelectasis of the lower lobe, of the left lung. The child was placed in an oxygen tent until the foreign body could be removed bronchoscopically. The bronchoscopic procedure was done on the evening of admission, and the screw, point up and partially covered with granulations, was removed. The following day roentgenograms revealed complete reinflation of the lower lobe of the left lung.

On the fourth day the patient again became acutely ill; stridulous breath sounds were present; the head was retracted, and indrawing of the suprasternal notch and the costal margins was noted. Breath sounds were suppressed over the lower lobe of the right lung, but no dulness was elicited. Pneumothorax was not demonstrated clinically. Tracheotomy was performed, a low incision being made and a no. 3 Jackson tube inserted.

The child improved after tracheotomy. Respirations became less labored, and good entry of air was present over both sides of the chest. On the fifth day, however, breath sounds were diminished or absent over the right side of the chest, which was hyperresonant to percussion. Roentgenograms demonstrated pneumothorax on the right, with the right lung collapsed to approximately one-half its normal size. The left lung was clear. A small amount of air was present in the subcutaneous tissues of the supraclavicular area. Since the patient evidenced no respiratory embarrassment, the pneumothorax was not treated. Convalescence was uneventful. The tracheotomy tube was removed on the fourteenth day. Roentgenograms at that time showed complete reexpansion of the right lung and normal aeration of the lower lobe of the left.

CASE 2.—D. K., a 3 month old white boy, was admitted to Children's Hospital of Michigan on Aug. 19, 1937, with respiratory difficulty. The child had been well until three weeks before admission, when a "cold" developed. He exhibited some periodic shortness of breath, which became progessively worse.

Examination showed a poorly nourished, poorly developed child, with clinical signs of rickets. He was not acutely ill. Moderate suprasternal retraction, dulness over the right subscapular region and fine rales in both pulmonary fields were noted. Roentgenograms showed an unclassified shadow of increased density opposite the right hilus, which was considered an enlarged gland, a pneumonic process or a tumor. The calcium and the phosphorus content of the blood were

5.6 and 1.2 mg., respectively, per hundred cubic centimeters. Calcium therapy was promptly started, and ten days later the calcium content of the blood was 12.0 mg. and the phosphorus content, 5.3 mg.

The child, however, showed signs of increasing dyspnea and greater retraction of the suprasternal notch and the intercostal spaces. A low tracheotomy was performed to relieve obstruction. No unusual complication occurred during the operation. Great relief was not obtained by the tracheotomy. Check-up roentgenograms showed the same unclassified mass opposite the right hilus as previously noted and in addition a limited amount of air in both the right and the left pleural cavity. An oxygen tent was used, but peripheral cyanosis, convulsions and an elevation of temperature to 106 F. ensued. The infant died one day after tracheotomy.

Autopsy showed a congenital stenosed larynx, with a threadlike glottic aperture. The unclassified mass seen in the roentgenograms proved to be the thymus, which

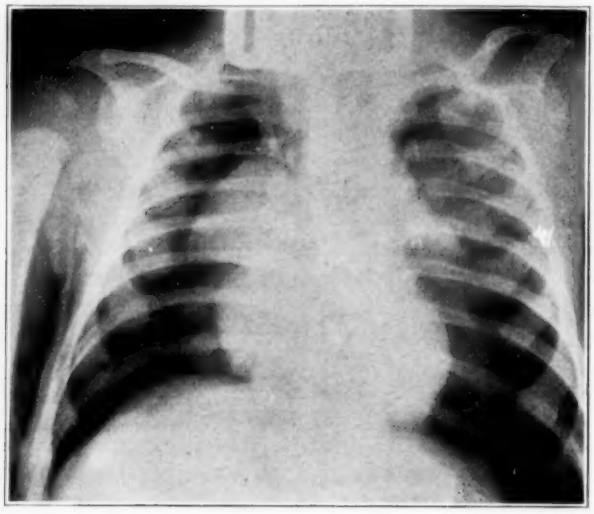

Fig. 1 (case 2).—Limited bilateral pneumothorax after tracheotomy. An enlarged mass opposite the right hilus which proved at autopsy to be thymus is shown.

weighed 11.5 Gm. and extended over the pericardium in the region of the middle lobe of the right lung. There was a large quantity of air about the posterior mediastinum, with large air-containing blebs which extended upward to surround the trachea. Limited pneumothorax was present on the right. The bronchi contained a mucosanguineous exudate; the lungs were atelectatic in the upper lobes and emphysematous elsewhere.

CASE 3.—D. K., a 2 year old white girl, was admitted to Children's Hospital of Michigan on Feb. 24, 1938, in a moribund condition. She was markedly cyanotic and showed the signs of air hunger. She had been well until six days prior to admission, when measles developed. Her condition was not critical until the day of admission, when she showed difficulty in breathing and became unconscious.

Examination showed a comatose child; the head was retracted and the respirations rapid, irregular and labored. Inspiratory stridor and marked retraction of the sternum, the costal margins and the suprasternal regions were present. The skin was dusky and covered with a confluent morbilliform rash; the lips and nail beds were deeply cyanotic. The chest was slightly hyperresonant, with fine rales and rhonchi over the base of the left lung posteriorly. There was no cardiac displacement.

The child was placed in an oxygen tent and given 5 minims (0.31 cc.) of solution of epinephrine hydrochloride, with no improvement. A low tracheotomy was performed one hour after admission. The child showed immediate improvement; the respirations became less labored; the cyanosis disappeared, and the child regained consciousness. Shortly afterward, moderate subcutaneous emphysema was noted over the upper part of the thorax.

The condition remained satisfactory for the ensuing four hours, when the patient again became markedly cyanotic. The respirations became noisy and labored. The chest was hyperresonant throughout; breath sounds were diminished or absent. Thoracic aspiration of an undetermined amount of air did not relieve the patient, and she died at 5 a. m., five hours after tracheotomy.

Autopsy revealed a markedly emphysematous mediastinum, with numerous large subserous air-containing blebs. Bilateral pneumothorax was present, with the lungs collapsed to about one-third their normal size. Areas of consolidation were present in the upper lobe of the right lung. The pulmonary parenchyma was atelectatic and congested elsewhere. A necrotic membrane covered the larynx, the trachea and the main bronchi.

CASE 4.—L. C., a 2 year old white boy, was admitted to Children's Hospital of Michigan on April 27, 1938, with elevation of temperature and difficulty in breathing. He had an infection of the upper part of the respiratory tract and during the six hour interval prior to admission had shown increasing respiratory embarrassment. He had difficulty in swallowing fluids.

Examination showed a well developed, acutely ill child with marked inspiratory stridor and indrawing of the suprasternal and the intercostal region. Cyanosis was not present. The throat was markedly injected and the tonsils hypertrophied, but no exudate was noted. The chest was clear except for occasional rales. A blood count showed the following values: hemoglobin, 14 Gm.; red cells, 5,100,000, and white cells, 13,200, with 92 per cent polymorphonuclears and 8 per cent lymphocytes.

The child was placed in a steam room, and sulfanilamide in doses of 1 Gm., with equal amounts of sodium bicarbonate, was given every six hours. He was restless, struggling for air, and tracheotomy was considered advisable. Prior to incision of the pretracheal fascia, a protrusion of pleura was noted in the region of the sternal notch. A hissing sound of rupture was heard. Incision of the third and fourth tracheal rings was performed and a no. 2 Jackson tube inserted. Thick pus was aspirated from the trachea, culture of which showed Streptococcus viridans.

Immediate improvement followed tracheotomy. The retractions ceased, and the child became quiet. Good entry of air was present over both sides of the chest, but some dulness to percussion was elicited over the right side anteriorly. Fluoroscopic examination showed a small, circumscribed area of decreased density overlying the left cardiac border. An area of increased density was present in the right lung near the hilus. No mediastinal shift or changes in normal diaphragmatic excursions were noted.

The child was kept in the steam room after tracheotomy, and sulfanilamide was administered every six hours. Roentgenograms taken the morning after tracheotomy showed air in the subcutaneous tissues of the neck, a limited amount of air about the mediastinum and an area of consolidation in the right lung opposite the hilus. Five days after tracheotomy, the infection had subsided sufficiently so that the tracheotomy tube could be blocked for half-hour trials. After a three day period of complete occlusion, decanalization was allowed. The subsequent course in this case of acute laryngotracheobronchitis was uneventful, and the child was discharged from the hospital on May 19.

CASE 5.—D. T., a 1 year old white boy, was admitted to Harper Hospital on Nov. 9, 1938, with labored breathing and attacks of cyanosis. Two months previous to admission, the child had received two immunizing doses of diphtheria toxoid. Elevation of temperature, a hoarse croupy voice and spells of convulsive coughing were present for several days.

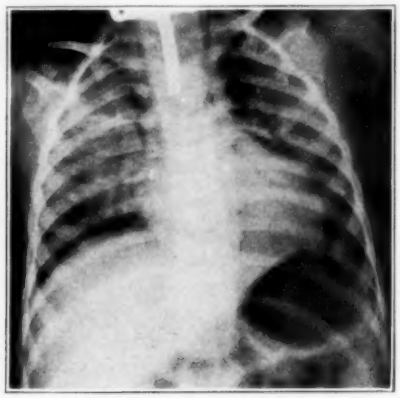

Fig. 2 (case 4).—Roentgenogram taken eight hours after tracheotomy, showing air in the subcutaneous tissues of the neck, a limited amount of air about the mediastinum and an area of consolidation in the right lung opposite the hilus.

Examination showed an acutely inflamed throat, with enlarged tonsils; no membrane was present. Inspiratory stridor with moderate retraction of the entire thoracic cage, unaccompanied by cyanosis, was noted. No relief was obtained by oxygen. A blood count showed the following values: hemoglobin, 12 Gm.; red cells, 5,200,000, and white cells, 10,800, with 62 per cent polymorphonuclears, 36 per cent lymphocytes and 2 per cent monocytes. Roentgenograms showed no evidence of parenchymal involvement of the lungs. A diagnosis of acute laryngotracheobronchitis was made.

The child continued to struggle and showed signs of increasing air hunger. A low tracheotomy was performed two hours after admission. The child struggled during the operation, and prior to incision of the trachea a portion of the right pleura protruded into the wound. A definite hissing sound of pleural rupture was heard. The tracheotomy was completed and immediate relief obtained by the insertion of the tube. Good entry of air in both sides of the chest resulted, and retractions ceased. No appreciable changes in resonance and respiratory sounds were elicited during the next two hours. Six hours after the tracheotomy, however,

the child's condition again became critical. Cyanosis was marked, and increased resonance was elicited over both sides of the chest with little air entry. A diagnosis of bilateral pneumothorax was verified by examination with the portable x-ray apparatus. By aspiration 175 cc. of air was removed from each side of the chest. The patient showed some improvement after this procedure. Air entry was good in the right side of the chest and less adequate in the left. The improvement was temporary, lasting about six hours. The signs of pneumothorax with pulmonary collapse were again present. The right pleural cavity was aspirated of 125 cc. of air and the left of 150 cc., with considerable relief.

A transfusion of 100 cc. of citrated blood was given, and fluids were administered parenterally. Sulfanilamide therapy was started. During the night following tracheotomy, the child showed some improvement; respirations decreased from 75 to 40 per minute. On the second day, however, air hunger was again present. There was a limited excursion of the left side of the chest, which was hyperresonant; 330 cc. of air was obtained from the left pleural cavity and 50 cc. from

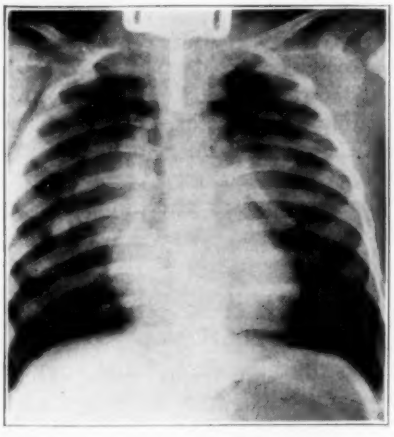

Fig. 3 (case 5).—Bilateral pneumothorax following rupture of the right pleural dome during tracheotomy.

the right. After this procedure, roentgenograms, taken with the portable apparatus, showed reinflation of both lungs, with limited pneumothorax in the right side of the chest and a discrete parenchymal consolidation throughout both lungs, suggesting congestion incidental to the collapse. Marked dyspnea continued, and the child died on the second day.

Autopsy showed a small amount of air in the left side of the chest. There was no emphysema about the trachea or larynx. The anterior pericardium contained numerous thin-walled subserous air blebs. The lungs showed early stages of consolidation, most marked in the upper lobe of the left. Along the large pulmonary vessels, massive air-containing blebs were observed. The larynx was injected and edematous, especially in the subglottic region.

Case 6.—L. T., a 13 month old Negro boy, was admitted to Children's Hospital of Michigan on Jan. 12, 1936, with a questionable history of having aspirated a bead. He had been playing on the floor the day of admission, when he suddenly started to vomit and gag. He became cyanotic and later had an emesis of sanguineous material.

Examination showed a child who was not acutely ill. The chest showed some decreased excursion on the right side, with flattening of the interspaces. The lungs were clear except for decreased respiratory sounds over the right side of the chest anteriorly. Roentgenograms of the chest showed an opaque beadlike foreign body in the right main bronchus below the bifurcation. The right lung was hyperventilated. Bronchoscopic inspection was performed on the day of admission, but the foreign body was not localized. Laryngeal edema and obstruction followed the procedure, necessitating tracheotomy. Low tracheotomy was performed with immediate relief.

Roentgenograms taken two days later visualized the foreign body in the right middle lobe bronchus. Bronchoscopic procedure was again unsuccessful. Dyspnea and respiratory embarrassment soon developed. Pneumothorax on the right was evidenced by the increased resonance, the mediastinal shift to the opposite side and the absence of respiratory sounds. Roentgenograms confirmed the diagnosis of pneumothorax on the right; the position of the bead was only slightly changed. The child showed signs of increasing air hunger and cyanosis. To relieve the pneumothorax, a no. 14 French closed tube catheter was inserted by means of a trocar into the right pleural cavity, the distal end being placed under fluid. A gush of air escaped and was followed by a succession of air bubbles. The boy's condition improved and remained satisfactory. Roentgenograms taken on the day following drainage of the pneumothorax demonstrated almost complete reexpansion of the right lung. The closed drainage of the right pleural cavity was discontinued after five days. Under fluoroscopic control, the foreign body was then removed. The subsequent course was uneventful. The tracheotomy tube was removed two weeks after admission, and the child was soon discharged.

CONCLUSIONS

Pneumothorax and mediastinal emphysema following tracheotomy are more common than the literature would suggest. The mechanisms involved and the probable modes of the entry of air into the thoracic cavity are discussed.

Six cases of acute laryngeal obstruction, with a mortality of 50 per cent, are presented. Pneumothorax and mediastinal emphysema in 5 cases followed tracheotomy. A case in which aspiration of a foreign body requiring tracheotomy was complicated by the development of tension pneumothorax after bronchoscopic procedure also is included. Of the group, 2 patients recovered with no primary treatment of the pneumothorax and 1 with continuous closed drainage; 1 died after a single inadequate pleural aspiration, 1 after repeated pleural aspirations and 1 without pleural aspiration.

Recognition and early adequate treatment of these conditions is essential. Continuous closed drainage obviates the objectionable features of repeated pleural aspirations. Great changes in thoracic and mediastinal pressure often prove fatal. The introduction of the bronchoscope prior to tracheotomy would eliminate many of the untoward complications mentioned. The prompt use of roentgenograms as an adjuvant to careful clinical interpretation is advocated.

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Case Reports

HEMOLYTIC STREPTOCOCCIC MENINGITIS OF OTITIC ORIGIN IN A YOUNG WOMAN AT TERM

Recovery and Delivery of a Living Child

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In the last few years, with the advent of sulfanilamide therapy, the mortality from meningitis due to the hemolytic streptococcus has fallen remarkably, and the literature abounds with reports of veritable series of cures. The following case is reported, therefore, not as a single case of meningitis with recovery but because of the added interest of recovery from the condition of a woman at term who at the height of her infection was delivered of a normal living baby—purely and simply another victory for sulfanilamide.

REPORT OF CASE

A woman aged 19, who was entering on the last few weeks of her pregnancy, was admitted to the Wilkes-Barre General Hospital late in the evening of July 3, 1938. She had a discharging ear. The discharge had begun five weeks before when the ear was opened because of an abscess of the middle ear which had resulted from insufflation of saline solution for a cold about three days earlier. Her medical history prior to that was irrelevant. She complained on admission of an excruciating left-sided headache, which had been getting worse for the past three hours and was unrelieved by the usual remedies. There was distinct nuchal rigidity, a bilateral Babinski reflex, an unimpressive Kernig sign and hyperactivity of the other reflexes. Her pupils were small, reactive and equal. There was no vomiting or blurring of vision. Her temperature was 100 F., her pulse rate 120 and her respiratory rate 26. There was but little discharge from the ear, and the condition of the drum appeared benign. There was a spot of tenderness far back over the mastoid area, with slight collapse of the posterior wall of the canal, but no supramastoid edema.

Immediate spinal puncture confirmed the diagnosis of meningitis, as will be noted in table 1, and extensive simple mastoidectomy was done. The only observations of note at the operation were that free pus was present at the tip, that the lateral sinus, though compressible, was covered with granulations and that the dura was not exposed. The wound was packed with iodoform gauze and closed. A piece of packing was likewise left in the external auditory canal. A culture of the pus from the tip was positive for hemolytic streptococci.

The temperature immediately rose to 101 F. and for the next two days varied between 104 (axillary) and 102 F. Administration of sulfanilamide was started immediately by mouth, 10 grains (0.65 Gm.), with an equal amount of sodium bicarbonate, every two hours, day and night. If the patient, because of her delirium, was unable to take the medication by mouth, 5 to 10 cc. of prontosil [1]

1. The disodium salt of 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′,6′-disulfonic acid. This is the substance previously known as prontosil soluble and now to be called neoprontosil.

TABLE 1.—*Analyses of Spinal Fluid*

Date	Cells	Polymorphonuclears	Lymphocytes	Globulin	Appearance	Pressure, Mm. of Hg	Amount Withdrawn, Cc.	Pressure After Withdrawal, Mm. of Hg	Organisms in Smears and Cultures
7/ 3	2,110	95	..	Strongly positive reaction	Cloudy	..	10	..	Streptococci or pneumococci on smear
7/ 4	7,300	90	..	Strongly positive reaction	Cloudy	..	25	10	Gram-positive diplococci
7/ 5	1,177	41	59	Faint trace	.....	..	30	10	Gram-positive cocci in a chain; hemolytic streptococci on culture
7/ 7	379	..	..	Negative reaction	.....	20	20	8	No free organisms but intracellular granular bodies of coccoid character in large numbers
7/ 8	111	..	..	Negative reaction	Clear	12	30	7	Gram-positive cocci, single, in pairs and in short chains (specimen had been in the ice box, so count is probably low)
7/ 9	50	..	..	Negative reaction	Clear	10	25	6	Culture negative on infusion broth and blood agar (specimen had been on ice by mistake)
7/11	38	..	..	Negative reaction	Clear	15	25	8	Culture negative
7/15	17	..	..	Negative reaction	.....	8	10	..	

was given intramuscularly. The dose was maintained for three days at 120 grains (7.78 Gm.) and then gradually reduced. On July 7 the concentration in the blood was 11 mg. in 100 cc. of blood. As the doses were reduced their amount was as follows: one day 90 grains (5.83 Gm.), one day 60 grains (3.88 Gm.), four days 40 grains (2.59 Gm.) and three days 30 grains (1.94 Gm.). Administration of the drug was then stopped altogether, on the thirteenth day of the patient's illness.

TABLE 2.—*Blood Counts*

Date	Sedimentation Rate	Hemoglobin, Gm.	Red Blood Count	White Blood Count	Polymorphonuclears	Eosinophils	Basophils	Large Lymphocytes	Small Lymphocytes	Mononuclears	Juveniles	Metamyelocytes	Myelocyte
7/ 4	.....	10.5 (66%)	3,400,000	12,050	70	..	..	3	15	5	7	..	..
7/ 5	0-13-19-20-21	11 (67.6%)	2,210,000	14,750	..	..	..	..	11	2	6	..	..
7/ 6	.....	10.5 (66%)	3,300,000	11,250	81	..	..	..					
7/ 7	.....	8.0 (51%)	2,400,000	9,900	..	..	..	4	12	4	21	5	..
7/ 8	.....	8 (50.6%)	2,470,000	10,050	54	..	..						
7/12	.....	9.5 (60%)	2,520,000	7,450	42	2	1	4	25	3	16	6	1
7/15	.....	9.75 (61%)	2,980,000	.....	..	..	..	..	..	..	..	..	
7/20	.....	10 (63%)	3,400,000	5,700	66	1	..	7	14	1	5	5	..
7/21	0-11-21-22-23												

TABLE 3.—*Transfusions*

Date	Type of Transfusion	Amount	
7/ 4	Plain transfusion	250 cc.	25 donors, 2 good, with 500 cc. 5% dextrose and saline solution
7/ 5	Plain transfusion	200 cc.	
7/ 6	Plain transfusion	200 cc.	
7/ 6	Immunotransfusion	200 cc.	12 donors, 1 good, with 500 cc. 5% dextrose and saline solution
7/ 6	Immunotransfusion	400 cc.	
7/ 8	Plain transfusion	600 cc.	20 donors, 1 good, with 500 cc. 5% dextrose and saline
7/ 9	Plain transfusion	200 cc.	13 donors, 3 good, with 500 cc. 5% dextrose and saline
7/10	Plain transfusion	200 cc.	
7/16	Plain transfusion	200 cc.	

The day after operation, arrangements were made for transfusions, and, as will be observed (table 3), plain transfusions and immunotransfusions were used frequently. The patient's blood was not typed, but, from the number of incompatibilities in the cross agglutination, it was probably of type 1.

Two days after admission she complained of abdominal pains and in the evening was delivered (with the aid of low forceps) of a normal boy, weighing 7 pounds 8¼ ounces (3,409.03 Gm.). Following her delivery there was profuse postpartum hemorrhage of about 600 cc. of blood. To combat this she was given another transfusion, and the uterus was packed. I was told that the baby at birth voided pink urine, which showed the presence of sulfanilamide in his system,

but my associates and I, because of our interest in his mother, lost the opportunity to make an interesting observation as to the concentration of the drug in his blood. After the first two days deep, severe jaundice, which lasted eight days, developed in the child, but he was otherwise unaffected by his (or his mother's) experience. He lost 8½ ounces (240.97 Gm.) in the first four days and then began to gain. At discharge (18 days old) he weighed 7 pounds 8½ ounces (3,416.11 Gm.).

After her confinement the mother's condition continued to improve. (A rise in temperature on the fourth day was considered due to absorption from the relaxed uterus.) On the seventeenth postoperative day she was allowed to be out of bed in a chair, and she was discharged on the twenty-first day, with her ear dry and her hearing for the whispered voice fairly good. She has been seen within the past few weeks; her ear remains dry, and she has no headaches. Her hearing is still slightly impaired, but she volunteered the information that it is gradually improving.

ACUTE LARYNGOTRACHEOBRONCHITIS

W. A. Cassidy, M.D., Omaha

Cases of fulminating laryngotracheobronchitis have been reported with considerable frequency, and the problems pertaining thereto have been thoroughly discussed by numerous practitioners. The attending difficulties and the seriousness of the situation are well recognized. The mortality, particularly of infants, is high, and without doubt it is impossible to save life in many instances. In consideration of these facts, the fortunate outcome in the case to be reported seems to justify its description. There is no doubt in my mind that without adequate facilities, competent nursing and the untiring efforts of all those concerned a successful termination could not have been brought about.

REPORT OF A CASE

S. H., a girl aged 11 months, was admitted to the Nebraska Methodist Hospital on May 28, 1936. Hospitalization was necessitated by stridor of gradually increasing intensity.

For a week before admittance to the hospital the child had shown symptoms of an acute infection of the upper part of the respiratory tract, of an afebrile type. During the two days prior to hospitalization a croupy cough developed, with inspiratory dyspnea.

On admission to the hospital, physical examination showed a well nourished child whose only apparent symptom was difficulty in obtaining adequate air. There was definite suprasternal and epigastric retraction on inspiratory effort. There was no cyanosis, but restlessness was noticeable. The nose contained a moderate amount of thin gray secretion. The pharynx was red, but no exudate or false membrane was present, and apart from edema the larynx was normal. Physical examination otherwise showed normal conditions, and data obtained by routine laboratory procedures were informative only in a negative way.

The child was placed in an oxygen tent, which operated continuously. During the succeeding twenty-four hours stridor was less troublesome, but it gradually became more apparent and more alarming until the evening of May 30, when it was evident that additional measures to supply oxygen would be necessary. Tracheotomy was performed with the patient under local anesthesia, with prompt subsidence of the dyspnea. Inspection of the interior of the trachea showed the mucosa to be inflamed, but no secretion or false membrane was evident. Other than greater edema, the larynx showed no specific pathologic change. Cultures taken from the interior of the airways produced no specific organism other than Bacillus coli, the presence of which was assumed to be the result of aspirating regurgitated food.

The case here reported was presented at a meeting of the Omaha Mid-West Clinical Society, Oct. 21, 1937.

Relief following tracheotomy was transient, in spite of continued use of the oxygen tent. Efforts to improve aeration by means of aspiration of the trachea through the tracheotomy tube gave only temporary relief. Eventually tracheal aspiration produced so little improvement that bronchoscopic inspection through the tracheotomy wound was decided on and carried out at 3 p. m. on May 31. The tracheal mucosa was found to be dehydrated and covered with thin, dry secretion. At the bifurcation was a large mass of inspissated yellow secretion, which was removed with forceps. Each main bronchus revealed the same appearance as the trachea. Physiologic solution of sodium chloride containing a small amount of 1:1,000 solution of epinephrine was introduced through the bronchoscope and the excess promptly aspirated.

Following the bronchoscopic treatment, the child experienced twenty-four hours of comparative comfort, but shortly thereafter the symptoms rapidly recurred, and the use of the bronchoscope was again required for the removal of obstructing secretion.

As will be seen from the accompanying table, bronchoscopic aspiration to relieve cyanosis and dyspnea assumed a prominent part in maintaining life.

Number of and Interval Between Bronchoscopic Procedures

	1	2	3	4	5	6
May 31.....	3:00 p.m.					
June 1.....	6:00 p.m.					
June 2.....	2:30 a.m.	9:30 a.m.	4:00 p.m.	8:00 p.m.		
June 3.....	1:00 a.m.	8:30 a.m.	10:30 a.m.	3:15 p.m.	7:15 p.m.	11:00 p.m.
June 4.....	4:00 a.m.	8:00 a.m.	12:00 noon	3:15 p.m.	9:00 p.m.	
June 5.....	1:40 a.m.	6:00 a.m.	11:30 a.m.	3:40 p.m.	9:40 p.m.	
June 6.....	3:45 a.m.	10:30 a.m.	2:30 p.m.	6:00 p.m.		
June 7.....	2:00 a.m.	6:15 a.m.	11:30 a.m.	6:30 p.m.		
June 8.....	1:00 a.m.	7:30 a.m.	1:00 p.m.			
June 10.....	8:15 a.m.	8:45 p.m.				
June 11.....	4:30 p.m.					
June 12.....	4:00 p.m.					
June 22.....	11:15 a.m.					

Bronchoscopic procedure was deferred each time as long as possible and resorted to only when the patient's condition was alarming and the clearing of the airway was imperative. On each occasion prompt relief was obtained, but recurring cyanosis and restlessness necessitated repeated use of mechanical means to clear the airways, until a total of thirty-seven aspirations was done over a period of thirteen days, before dyspnea ceased. The thirty-eighth bronchoscopic procedure and direct inspection of the larynx was done ten days after the cessation of active symptoms to satisfy me that it was safe to remove the tracheotomy tube permanently. The child was dismissed from the hospital on June 25 in good condition.

In addition to bronchoscopic treatment, the child received pediatric care of great importance. The problem was complicated by complete anorexia. Gavage was necessary for twelve days, and on two occasions liquids were administered intraperitoneally. Throughout the period of active symptoms the child was removed from the oxygen tent only when bronchoscopic treatment became necessary. Efforts were made to keep the air within the tent as nearly saturated with moisture as possible. The use of vaporizers and other methods were adopted to keep the air in the room extremely moist, so that the intake of the tent would carry as much moisture as could be supplied. Small quantities of physiologic solution of sodium chloride containing a few drops of 1:1,000 solution of epinephrine were instilled through the tracheotomy tube at frequent intervals. During the last few days small quantities of glycerin were added to the solutions

instilled into the trachea, in the hope that an outpouring of thinner secretion might be stimulated. My associates and I believed that there was a rather favorable response to this medication. On June 2, 1936, at the suggestion of the radiologist, 80 roentgen units of radiation, filtered through the canvas of the oxygen tent, was administered over the chest. No demonstrable improvement in the child's condition followed the radiation therapy.

Roentgenograms of the chest showed the extent to which pulmonary function was compromised. The first roentgenogram, made on June 1, 1936, was interpreted as follows: Parietal pneumothorax was present on the right. The middle lobe of the right lung was completely collapsed, possibly because of bronchial occlusion; there was a slight bilateral perihilar inflammatory reaction. The second, made on June 6, revealed nearly complete expansion of the right lung since the previous examination. No atelectasis of the middle lobe of the right lung was suggested at that time. The right perihilar shadows showed a fuzzy thickening, interpreted as a peribronchial pneumonic reaction of slight degree, with no evident area of confluent consolidation through either lung. The pleural sacs appeared clear.

A survey of the literature of the subject indicates that this case is unique in the frequency of the fulminant attacks and the number of bronchoscopic aspirations necessary to effect a cure. The next largest number undertaken in a single case, seventeen, was reported by Clerf [1] in 1924. His case differs, however, from the one just described in that the problem developed subsequent to the removal of a foreign body.

CONCLUSION

The experience just described emphasizes the importance in such cases of hospitalization in an adequately equipped institution, as insisted on by Gittins,[2] Lyman Richards,[3] H. L. Baum [4] and others.

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INFLUENZAL MENINGITIS WITH RECOVERY

Treatment with Mastoidectomy, Sulfanilamide and Continuous Spinal Drainage

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Influenzal meningitis remains a disease with a high mortality, which has been only slightly lowered by the use of specific serum.[1] A few reports are now appearing in the literature on treatment with sulfanilamide, but these, in general, have been discouraging.

Long and Bliss [2] stated that sulfanilamide inhibits the growth of Haemophilus influenzae in vitro but that its use in the treatment of clinical meningitis was much less successful. McIntosh, Wilcox and Wright [3] reported treatment in 2 cases with anti-influenza serum and sulfanilamide. They stated that the use of this drug was of questionable value in their cases. Basman and Perley [4] reported 1 case of fatal influenzal meningitis treated with sulfanilamide, but the infection was considered to be overwhelming. McQuarrie [5] described treatment of influenzal meningitis with anti-influenza serum and sulfanilamide. His patient died after a protracted illness, despite some clinical improvement following the use of sulfanilamide. Taylor [6] reported a fatal case in which sulfanilamide had given no clinical improvement. Young and Moore [7] are the only authors discovered in a recent review of the literature to report a case with recovery. Their patient was treated with both anti-influenza serum and sulfanilamide.

Recently we have had a patient with influenzal meningitis who recovered after treatment with mastoidectomy, prontosil [8] solution, sulfanilamide by mouth and continuous spinal drainage, in addition to customary supportive treatment. No specific serum was used.

From the Departments of Pediatrics and Otolaryngology of the Medical School, Ohio State University, and the Children's Hospital.

1. Silverthorne, N.; Fraser, D. F., and Snelling, C. E.: J. Pediat. **10**:228, 1937.

2. Long, P. H., and Bliss, E. A.: Para-Amino-Benzene Sulfonamide and Its Derivatives: Experimental and Clinical Observations on Their Use in the Treatment of Beta-Hemolytic Streptococcic Infection; Preliminary Report, J. A. M. A. **108**:32 (Jan. 2) 1937.

3. McIntosh, R.; Wilcox, D. A., and Wright, F. H.: J. Pediat. **11**:167, 1937.

4. Basman, J., and Perley, A. M.: J. Pediat. **11**:212, 1937.

5. McQuarrie, I.: J. Pediat. **11**:188, 1937.

6. Taylor, H. W.: Arch. Pediat. **55**:131, 1938.

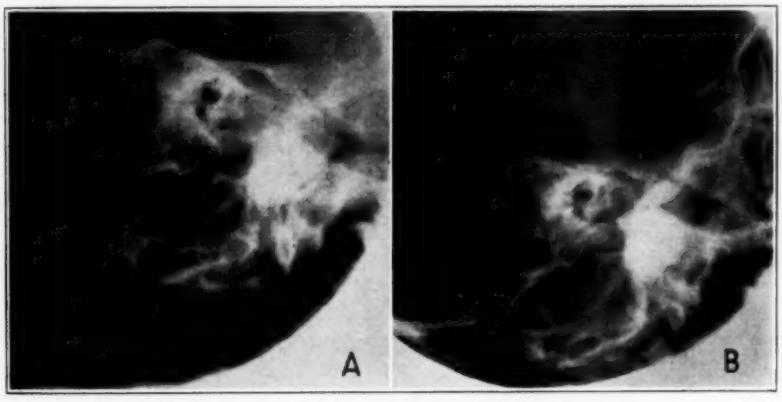
7. Young, R. H., and Moore, C.: Arch. Pediat. **55**:282, 1938.

8. The disodium salt of 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′,6′-disulfonic acid. This is the substance previously known as prontosil soluble and now to be called neoprontosil.

REPORT OF A CASE

History.—C. K., a 6 year old white boy, was referred to us on Feb. 23, 1938, by Dr. Henry Karrer of Dublin, Ohio, with a diagnosis of acute mastoiditis and questionable meningitis. The parents stated that measles had developed on Feb. 1, 1938, twenty-three days previous to admission. The course of the disease had been uneventful until February 18, five days before admission, at which time the patient had become feverish and complained of anorexia and headache. There had also been slight drainage from both ears. Two days before coming to the hospital the patient had had slight stiffness of his neck and had vomited several times. There had been no chills, tremors or convulsions. The personal and family history were not contributory.

Examination.—The patient was an acutely ill, pale, dehydrated, malnourished white boy, who responded feebly to questions. His weight was 40 pounds (18.1 Kg.). The temperature was 103 F. per rectum. The pulse rate was 96 per minute and of good quality. The respiratory rate was 24; respiration was regular



Roentgenograms of the right (*A*) and the left (*B*) mastoid, taken on Feb. 23, 1938.

and of normal depth. The scalp and eyes were normal. The right tympanic membrane was covered with exudate and bulging. The left drum was dull and retracted. The right mastoid was tender but not swollen. No abnormal mastoid signs were made out on the left. The pupils were equal and reacted to light and in accommodation, and extraocular movements were normal. Ophthalmoscopic examination showed no abnormality. A mucopurulent discharge was noted on the floor of each nasal cavity. The mouth revealed some dental caries. The pharynx was injected. The tonsils were inflamed and hypertrophied and showed a slight exudate. The neck was moderately stiff, and there was bilateral cervical lymphadenopathy. The lung fields were clear and the heart sounds normal. The abdomen was soft, but there was a diffuse tenderness, more marked in the epigastrium. The genitalia and extremities were normal. The neurologic examination showed normal tendon reflexes, negative Babinski and positive Kernig and Brudzinski signs. The abdominal and cremasteric reflexes were absent. A roentgenogram of the mastoids taken on February 23 was interpreted by Dr. Huston F. Fulton as follows: "Both mastoids are in a pathologic condition. The disease is further advanced on the right side."

Laboratory Observations.—The Kahn test of the blood gave negative results. The patient's blood was type II, Moss. The discharge obtained from the right ear on February 23 produced a staphylococcus on blood agar culture. A culture of blood drawn on February 24 was sterile. A culture of material from the throat was reported on February 25 to contain a staphylococcus. A test of the fragility of the blood showed hemolysis to begin with a 0.34 per cent solution of sodium chloride and to be complete with a 0.28 per cent solution. The urine at five routine examinations was essentially normal. Culture of the pus found in the right mastoid at operation showed gram-negative pleomorphic organisms.

Course in the Hospital.—The patient was admitted at 1:30 a. m. on Feb. 23, 1938. A bilateral myringotomy was done. A seropurulent discharge was obtained

TABLE 1.—*Analysis of the Spinal Fluid*

Date	Appearance	Cells	Lymphocytes, %	Polymorphonuclears, %	Sugar Reaction	Globulin Reaction	Smear of Sediment	Growth on Culture
2/23	Cloudy	850	5	95	Negative	Positive	H. influenzae	
2/24	Turbid	3,000	6	94	Negative	Positive	H. influenzae	H. influenzae
2/25	Turbid	...	..	..	.....	.....	.....	H. influenzae
2/26	Ground glass	370	..	..	Negative	Positive	H. influenzae	
2/27	Clear	120	..	..	Negative	Positive	Negative	
3/ 3	Clear	50	60	40	Negative	Positive	Negative	None
3/ 7	Clear	80	75	25	Positive	Negative	Negative	None
3/11	Clear	50	..	..	Positive	Negative	Negative	None

TABLE 2.—*Examination of the Blood*

Date	Hemoglobin, %	Erythrocytes	Leukocytes	Segmented Cells, %	Stab Cells, %	Juveniles, %	Lymphocytes, %	Eosinophils, %
2/23.....	60	3,890,000	18,800	70	4	1	25	..
2/26.....	58	3,140,000	22,400	78	1	2	19	..
3/ 3.....	73	2,840,000	11,600	67	8	..	25	..
3/10.....	98	4,620,000	7,900	50	6	..	42	2

from the right ear. The child took small amounts of water by mouth and rested fairly well during the remainder of the night. A lumbar puncture at 8 a. m., as has been reported, revealed pleomorphic gram-negative organisms in the smear.

A simple mastoidectomy was performed on the right on the evening of February 23. The mastoid showed subcortical cells, diploic and semisclerotic in type; the periantral cells were necrosed, and the antrum was filled with granulations and purulent exudate. A small area of dura over the middle fossa was exposed, as was a segment of the lateral sinus; both appeared normal.

On February 25 a large lumbar puncture needle was inserted into the fourth interspace, with the patient in a position of normal extension, and continuous spinal drainage was attempted. The needle was held in position by means of small forceps, which were firmly strapped to the back by adhesive tape. The

patient was placed on a Bradford frame, with the needle draining into a receptacle below. In the hope of increasing the production of cerebrospinal fluid and improving the quantity of drainage, a continuous intravenous drip of hypotonic saline solution was given at the same time. A 0.6 per cent solution was used, as this was thought to be far enough above the point of fragility of the red blood cells (0.34 per cent) to be safe and yet was definitely hypotonic. Four ounces (118 cc.) only of spinal fluid were obtained in the six hours after the beginning of the intravenous drip, at the end of which the sudden development of a shaking chill, cyanosis and a weak, thready pulse prompted the discontinuance of both the spinal drainage and the administration of hypotonic saline solution. On the next day, however, continuous spinal drainage was again started, but 5 per cent dextrose in physiologic solution of sodium chloride was employed in the constant intravenous drip. Slightly over 20 ounces (591 cc.) of cerebrospinal fluid was obtained in the succeeding seventy-two hours, before the needle was removed. It was in this period that marked clinical improvement, with a striking drop in pleocytosis, occurred.

From the first day of hospitalization, the patient was given intramuscularly 10 cc. of protonsil [8] solution twice daily until he was able to take sulfanilamide by mouth. He was then given 2 Gm. per day in divided doses from February 27 until March 1. During the remainder of his stay in the hospital he was given 1.3 Gm. of sulfanilamide per day in divided doses. The patient was given also the usual forms of supportive treatment, such as subcutaneous and intravenous infusions, and three blood transfusions.

There were daily elevations of temperature to 104 or 105 F. during the first three days in the hospital. Thereafter, the temperature curve gradually fell and remained normal after the seventh day. The pulse rate, which was rapid when the temperature was elevated, remained within normal limits after the seventh day. The respiratory rate was approximately 20 per minute.

Convalescence.—At the time of discharge, on March 12, the patient's neurologic examination gave entirely normal results. The fundi were normal. The mastoidectomy wound was almost completely healed. Convalescence at home during the following three months was uneventful, and there has been no evidence of sequelae. The patient has been gaining weight and is enjoying all of the usual activities of a boy of his age.

COMMENT

It is generally accepted that influenzal meningitis is a primary disease not associated with any striking infection of the respiratory tract. In this case the symptoms referable to the ear preceded the meningeal signs, and typical pleomorphic organisms were cultured from both the mastoid secretion and the spinal fluid. Here, we feel, primary mastoiditis was responsible for the influenzal meningitis, and surgical removal of the septic focus was a most important part of successful therapy.

Continuous spinal drainage was successfully carried out on this patient and was likewise felt to be an important factor in the comparatively prompt recovery. Our experience with hypotonic saline solution was not encouraging, but adequate drainage fortunately was maintained with the more routine 5 per cent dextrose in saline solution, given in the form of a constant intravenous drip.

Prontosil [8] solution and sulfanilamide were employed in moderate dosage in this case. We cannot state definitely that chemotherapy had

any specific effect on the organism, but we do feel that it may have been of some benefit. Further study and clinical trial of this drug will establish its place in the treatment of influenzal meningitis. We do not feel that there were any untoward consequences from its use for this patient.

SUMMARY

A case of virulent influenzal meningitis with recovery is reported.

The condition was associated with influenzal mastoiditis, probably primary.

Treatment consisted of mastoidectomy, constant lumbar drainage and chemotherapy (prontosil [8] solution and sulfanilamide), in addition to ordinary supportive measures.

No specific serum was administered.

New Instruments

SELF-CLEANING GOGGLES FOR THE BRONCHOSCOPIST

KARL GRUPPE, M.D., UTICA, N. Y., AND
G. HARVEY CAMERON, PH.D.,* CLINTON, N. Y.

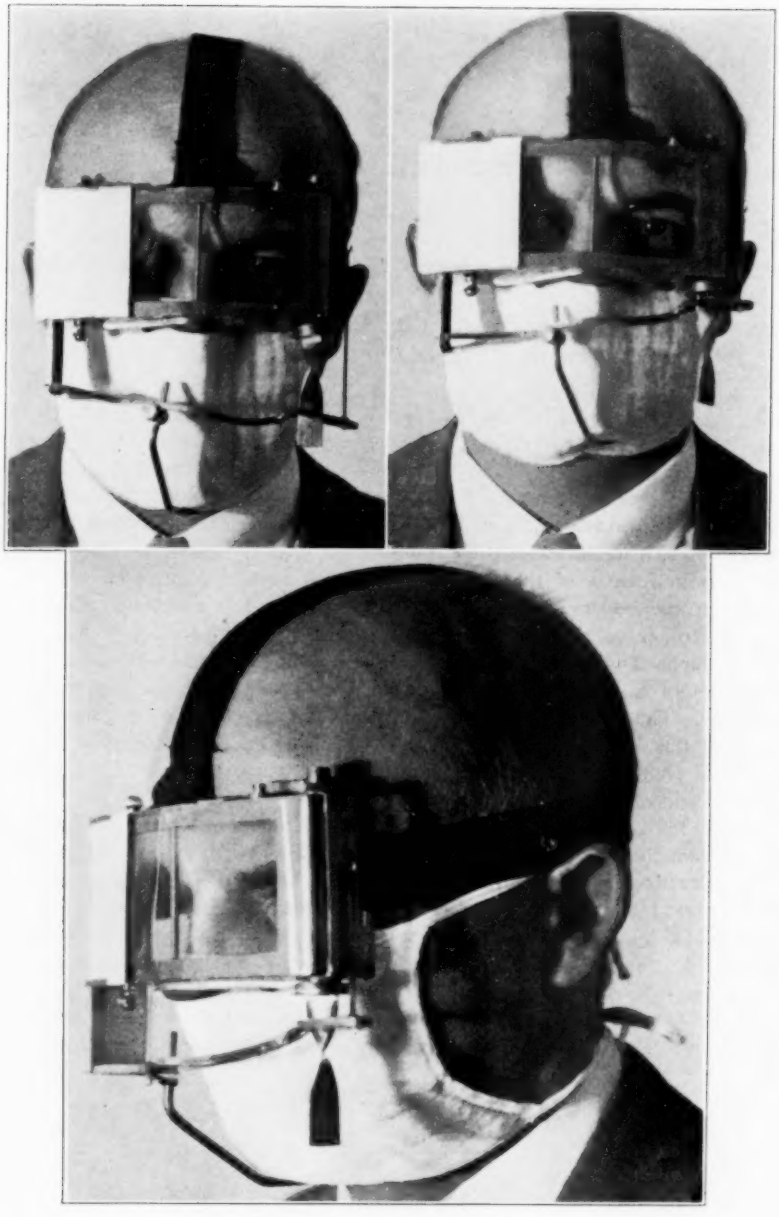
During the course of laryngeal or bronchoscopic examination of patients whose bronchial secretions are profuse, considerable annoyance and loss of time are occasioned by the frequency with which the examiner's protecting goggles become clouded and require cleaning. To obviate this time-consuming difficulty a pair of self-cleaning, or self-replacing, goggles was constructed, description and photographs of which we herewith present:

The main feature of the apparatus is a roll of clear, uncoated film support mounted on two rollers so placed in their supporting frame that the film, in winding from one roller to the other, passes across the line of vision. The receiving roller is supplied with a winding and ratchet mechanism actuated by down and up movements of a cross arm, which, in turn, is actuated by opening and closing of the jaw. That portion of the film which has not yet passed before the examining eye (with this instrument, the right eye) is protected from smearing by a piece of blotting paper. This is held in a grooved frame to permit its adjustment out to the margin of the line of vision.

In use, the instrument is satisfactory. The film permits clear vision and does not fog from the moist breath. As it becomes clouded with coughed-out secretion a clear area of film may immediately be placed before the examining eye by one downward motion of the jaw, and as the roller may hold as much as 4 feet (122 cm.) of film the entire examination may be conducted with perfectly clear vision. The instrument is readily slipped on over a mask and does not interfere with ordinary talking.

In the photographs the two front views show the cross bar down and up, respectively. The oblique view shows more detail of the winding mechanism.

* From Hamilton College.



Photographs of the instrument in use.

Abstracts from Current Literature

Ear

Contribution to the Study of Grippe Otitis, Myringitis Bullosa Hemorrhagica, and Its Relationship to Latent Scurvy. S. L. Ruskin, Laryngoscope **48**:327 (May) 1938.

Ruskin attributes myringitis bullosa haemorrhagica associated with grip to latent scurvy and recommends for its treatment parenteral administration of the calcium salt of vitamin C. This thought was prompted by Hess's description of infantile scurvy and infectious scurvy, a type of the disorder brought about by superimposing a secondary infection on the primary nutritional disturance. Hess noted an epidemic of grip that precipitated an epidemic of scurvy which was exceptional in its hemorrhagic tendency and which failed to respond to specific therapy by diet.

This therapeutic failure is explained by the present knowledge of the structure of ascorbic acid and its oxidation-reduction properties. The intestinal toxins react with it, depriving the body of normal absorption of vitamin C. Ruskin was prompted to secure the benefit of vitamin C therapy in these cases by parenteral administration of the remedy.

In the period of one year all patients (10) with myringitis bullosa haemorrhagica received large doses of vitamin C and 3 cc. of a 15 per cent solution of calcium ascorbate intramuscularly. Only a few of the patients received paracenteses, and all showed signs of improvement within twelve hours, the condition being resolved within four to five days. There was a marked lessening of pain after the first day, and the patients who showed a thin serous discharge from the middle ear received no medication to the ear and no irrigations. The cotton in the aural canal was changed whenever moist.

Wood, Newark, N. J.

Symposium. What is Justifiable to Do in Otitic Meningitis? A Brief (Historical) Review of the Operative Treatment of Purulent Meningitis. T. J. Harris, Laryngoscope **48**:458 (July) 1938. Surgical Indications in Suppurative Meningitis. J. G. Dwyer, ibid. **48**:461 (July) 1938. The Surgical Treatment of Meningitis. I. Friesner and H. Rosenwasser, ibid. **48**: 427 (July) 1938. Sulfanilamide and Related Compounds in the Treatment of Meningitis Secondary to Ear and Sinus Infections. E. Appelbaum, ibid. **48**:482 (July) 1938.

Harris describes the methods and procedures offered since 1873 for the surgical treatment of purulent meningitis of otitic origin.

Dwyer emphasizes the need for proper diagnosis of meningitis made essentially from an examination of the spinal fluid. He deals extensively with the interpretation of findings in the spinal fluid to determine exactly the type of meningitis, if any, that is present. He states that the surgical treatment of meningitis is that of the focus of infection. He recalls that a cloudy spinal fluid may be a tissue reaction to any irritant, including an infection. With no organism, the focus is still external to the meninges, and its surgical removal is strongly indicated. In the bacteriologic examination, it is suggested, a culture will be made more certain by planting 1 to 2 cc. of the centrifuged specimen.

Appended to the paper is a chart of the main points in the diagnosis of meningitis from the cerebrospinal fluid, as well as the questionnaire on a case of meningitis used by the New York Committee of the American Otological Society, Inc.

Friesner and Rosenwasser feel that the classification of meningitis into various forms according to the results of cytologic, chemical and bacteriologic examina-

tion is misleading and that the infection occurs in the tissues that comprise the meninges and not in the fluid itself. They say the changes in the fluid are merely expressions of the degree, severity, type and extent of the inflammation of the meninges and feel that the value of drainage of the fluid is theoretic. In discussing the surgical therapy they recommend:

1. For meningitis secondary to infection of the labyrinth, prompt labyrinthectomy with inspection of the dura of the middle and the posterior fossa, because the tendency for the infection to remain localized and heal is exceedingly slight.

2. For meningitis secondary to infection in the lateral sinus, an effort to get well beyond the limits of the disease, proximally and distally, laying the sinus open widely; if the bulb is thrombosed and purulent thrombi are present, drainage from the bulb by irrigation with Dakin's solution.

3. For meningitis secondary to infection in the middle ear, the mastoid and the petrous bone, after a complete mastoidectomy, a careful systematic search for leads into the petrous pyramid, the peribulbar cells and the zygoma by means of the Eagleton type of exploration until every pathway of extension has been carefully investigated, without opening the dura.

They conclude that the surgical treatment of meningitis is the early and adequate drainage of the focus from which the meningitis takes its origin.

In relating his experiences in using sulfanilamide in the treatment of otitic meningitis, Appelbaum states that smaller doses at more frequent intervals are more effective than larger doses at longer intervals, that the routine administration of sodium bicarbonate is advisable and that daily blood counts are important. In his series of 32 cases of pneumococcic meningitis 4 patients recovered. In the series of 26 cases of meningitis due to hemolytic streptococci 21 patients recovered and 5 died. He emphasizes the necessity of eradication of the primary focus and considers important the adequate drainage of the subarachnoid space by lumbar punctures (cisternal or ventricular if block occurs). He discourages radical methods of drainage, such as forced spinal or perivascular drainage.

In the discussion Eagleton recommended that after obliterating the primary focus of infection one open the cerebrospinal fluid system of the area where the infection entered, drain the infected fluid adjacent to the focus and then immediately close the dural opening with a piece of actively proliferating tissue, preferably mucous membrane of the lip or fascia lata. He feels that the cerebrospinal fluid adjacent to the focus of infection contains organisms in many cases in which the fluid drawn by lumbar puncture simply shows a high cell count.

Dr. Josephine B. Neal cautioned that cisternal puncture in cases of meningitis offers a different problem from cisternal puncture in cases of syphilis of the central nervous system because of the adhesions and congested blood vessels. She had heard of 25 or 30 fatalities in cases of meningitis, at the hands of experienced operators.

WOOD, Newark, N. J.

SCARLET FEVER COMPLICATED BY MASTOIDITIS WITH MENINGITIS AND SINUS THROMBOSIS WITH JUGULAR PHLEBITIS. T. B. LAYTON, Proc. Roy. Soc. Med. **31**:909 (June) 1938.

The case of a child of 6 who had earache but no discharge of pus is reported. Because of high fever and meningitic signs the mastoid was opened; two days later the lateral sinus was opened and a clot removed. This procedure was followed in two more days by ligation and opening of the jugular vein and evacuation of a clot, after which the child rapidly recovered.

CAMPBELL, Philadelphia.

SCARLET FEVER COMPLICATED BY MASTOIDITIS AND LATERAL SINUS THROMBOSIS. T. B. LAYTON, Proc. Roy. Soc. Med. **31**:910 (June) 1938.

A case is reported of a child of 5½ years in whom abscess of the middle ear developed during convalescence from scarlet fever. Operation disclosed an extra-

dural abscess and granulations over the lateral sinus. Six days later the sinus was opened and a clot removed. As the fever did not subside, six days later the jugular vein was ligated. After this procedure the child recovered quickly.

CAMPBELL, Philadelphia.

REPORT ON CEREBRAL ABSCESS OBSERVED AT THE EAR CLINIC IN AGRAM. A. SERCER, Monatschr. f. Ohrenh. **72**:946 (Oct.) 1938.

The writer reports 34 cases of otogenic cerebral abscesses observed at the clinic in Agram during the past sixteen years. On the basis of those cases the well known facts about the pathologic picture and clinical course of cerebral abscess are repeated and the literature quoted rather superficially. As far as the treatment is concerned, the writer mentions quite hypothetically that injection of drugs (rivanol) into the carotid artery may increase the local reaction of the brain tissue around the abscess and hinder the propagation of encephalitis.

LEDERER, Chicago.

INVOLVEMENT OF THE MASTOID AND GRANULOPENIA IN A CASE OF BRUCELLA INFECTION. A. OPPENHEIMER, E. W. DENNIS and M. BADEEN, Pract. oto-rhino-laryng. **1**:305 (Sept.) 1938.

The authors report a case of infection with Brucella involvement of the mastoid in which there was no clinical evidence of true mastoiditis but the diagnosis was made roentgenographically.

The patient recovered after a simple mastoidectomy and the institution of vaccine therapy.

PERSKY, Philadelphia.

NYSTAGMUS DUE TO OCULAR MOVEMENTS. ERICH RUTTIN, Pract. oto-rhino-laryng. **1**:314 (Sept.) 1938.

The author presents a case in which nystagmus and vertigo followed mastoidectomy. When the patient closed her eyes tightly and then opened them, on looking to the right she would show nystagmus to the right, and on looking to the left, nystagmus to the left. The author believes that this phenomenon was due to associated innervation of other branches of the oculomotor nerve with the levator palpebrae superioris. Since it occurred during her menses, he suggests that it may have an endocrine cause.

PERSKY, Philadelphia.

INTRAVENOUS ALCOHOLIC INJECTIONS IN A CASE OF MASTOIDITIS COMPLICATED WITH THROMBOSIS OF THE JUGULAR BULB. MINORU SASAKI and SEIICHI KAWATA, Oto-rhino-laryng. **11**:701 (Aug.) 1938.

The authors report the case of a 49 year old man, a school teacher, in whom otitis media on the right developed in January, becoming progressively worse, and who on April 3, three months after the onset, had a simple mastoidectomy. The smear from the swab was streptococcic, but the organisms failed to grow on culture. On the fifth postoperative day signs of pulmonary embolism on the right developed. On the sixth postoperative day a ligation of the jugular vein was made after the uncovering of mural thrombosis of the jugular bulb. After the second operation the septic temperature persisted for several days, and the patient appeared to be moribund in spite of transfusion and other supporting measures. As a last resort on the eleventh postoperative day 15 cc. of 33 per cent alcohol was slowly injected intravenously; 10 cc. was injected on the thirteenth and 15 cc. on the fourteenth postoperative day. After each injection the temperature rose sharply to nearly 104 F., but shortly thereafter fell to 100 F., and after the third injection the temperature dropped to subnormal and remained so for several days.

Intravenous injections of alcohol have been tried, by Landau in France in 1931 and by Orou in Germany in 1935, for the treatment of pulmonary abscess, septicemia and other conditions. More recently the Japanese have tried injections of alcohol in the treatment of influenza, pneumonia, erysipelas and osteomyelitis, with varying success. This is the first time that alcohol injection was given for sinus thrombosis. According to the authors alcohol stimulates the development of opsonin and bacteriolysin and thus favors the natural defense of the body.

HARA, Los Angeles.

Pharynx

THE INCIDENCE OF TONSILLECTOMY IN SCHOOL CHILDREN. J. ALISON GLOVER, Proc. Roy. Soc. Med. **31**:1219 (Aug.) 1938.

Tables are presented showing the rapid rise in vogue of the operation for the removal of tonsils and adenoids at the beginning of the twentieth century, the comparative slackening of interest during the years of the war, the high tide of 1931 and the subsequent ebb. From statistics presented, it appears that more boys are operated on than girls, and the highest incidence of the operation is between 5 and 7 years of age. Further tables and statistics show the incidence of the operation in the United States and in various sections of the British Isles and indicate that tonsillectomy is at least three times as common for the well-to-do as for the poorer classes and that the mortality from the operation is larger than is generally appreciated.

The subject is discussed by Mr. T. B. Layton, Sir Arthur MacNalty, Mr. E. D. D. Davis, Dr. Percy Stoks and Dr. R. R. Garrow, who express individual opinions on the incidence of the operation, the technic of operating, the symptomatology and other subjects.

CAMPBELL, Philadelphia.

NASAL DIPHTHERIA AND TONSILLECTOMY. JOSEPH BERBERICH, Pract. oto-rhino-laryng. **1**:311 (Sept.) 1938.

The author presents 5 cases of carriers of diphtheria in whom tonsillectomy has resulted in disappearance of diphtheria bacilli in the nose. In view of this finding he suggests that there may be some special anatomic connection between the nose and the tonsils.

PERSKY, Philadelphia.

Larynx

PARALYSIS OF BOTH RECURRENT LARYNGEAL NERVES DUE TO ECTASIA AND ANEURYSM OF THE AORTA. GIUSEPPE SALVADORI, Valsalva **14**:284 (June) 1938.

The author describes a case of paralysis of both recurrent laryngeal nerves due to an enormous aortic aneurysm, which pushed the trachea to the right, stretching the right recurrent laryngeal nerve, while the left was paralyzed by the ectasia of the whole arch of the aorta.

The patient died from rupture of the aneurysm into the trachea, and necropsy confirmed the diagnosis.

LUONGO, Philadelphia.

CYSTADENOMA LARYNGIS. E. WESSELY, Monatschr. f. Ohrenh. **72**:682 (July) 1938.

The author describes a cystadenoma of the larynx which perforated the hypothyroid membrane and was clinically diagnosed as a laryngeal cyst. The tumor was extirpated by an external operation, but about one year later a fibrosarcoma developed in the scar of the skin. The author believes that the cystadenoma originated from thyroid tissue which had been displaced in embryonic life.

LEDERER, Chicago.

CLINICAL OBSERVATIONS OF THE PATHOGENESIS AND COURSE OF HEMATOGENOUS LARYNGEAL TUBERCULOSIS. J. SPIRA, Monatschr. f. Ohrenh. **72**:700 (July) 1938.

Spira believes that hematogenous tuberculosis of the larynx has to be taken into consideration when the physical and the roentgen examination of the lungs yield negative results or that hematogenous tuberculosis of the lungs, not greatly advanced, must be borne in mind. From the clinical point of view he describes two forms of hematogenous tuberculosis of the larynx: The first, a malignant form, starts with intensive symptoms at the entrance of the larynx, spreading rapidly into the larynx itself. Mirror laryngoscopy reveals an acute swelling of the entrance into the larynx. The second form usually begins at the vocal cords as an infiltration, which later undergoes ulcerative changes. This form is more benign and generally heals after a shorter or longer period.

LEDERER, Chicago.

Nose

THE CROOKED NOSE. A. A. CENELLI, Laryngoscope **68**:760 (Oct.) 1938.

Cenelli devised a classification of the external nose into thirds and discusses, with photographic illustrations, the various plastic operations applicable to each. For correction of fractures of the upper third of the external nose he has substituted for the usual Joseph operation of removing a triangular segment from the broad aspect of the deviation a procedure consisting of a high resection of the dislocation of the septum caused by the fracture, surgical dislocation of the nasal bones from their lateral attachments and reposition in the midline.

MILLER, Philadelphia.

MALIGNANT TUMORS OF THE PARANASAL SINUSES. B. SZENDE, Monatschr. f. Ohrenh. **72**:925 (Oct.) 1938.

The writer describes his experiences in 70 cases of malignant tumor of the paranasal sinuses and emphasizes the well known fact that in many such cases no pathologic condition is discovered within the nose. He often finds exophthalmos, so that he believes that in every fifth case exophthalmos is of nasal origin. As far as the treatment is concerned, he does not ligate the external carotid artery in cases of tumor of the ethmoid sinus and always operates as radically as possible, carrying out the after-treatment with roentgen rays or radium.

LEDERER, Chicago.

CAUSATIVE FACTOR IN NASAL HEMORRHAGE. GÖSTA DOHLMAN, Acta oto-laryng. **26**:575, 1938.

Dohlman has attempted by microscopic study of the involved mucosa to find the cause of the repeated hemorrhages of the nose occurring at Kiesselbach's area, where the transition from pavement to ciliated columnar epithelium occurs. In general he concludes that they are due not to external traumatic agents, such as fingers, handkerchiefs and dry air, but to endogenous causes. He has found changes in the mucous membrane of this area which are similar to the changes produced by an eczematous process elsewhere, changes by which the area in question tends to become denuded of its epithelial covering. When this takes place, the capillary loops of the papillae lie close to the surface, unprotected by an epithelial covering. In his sections he found that these vessels were filled with thrombi, not only at the bleeding point but also some distance away. In 2 cases he was able completely to control repeated bleeding from this area by dietary changes, and in 1 of these the bleeding recurred a year later after a dietary indiscretion, to be again entirely controlled by strict adherence to the prescribed diet. His idea, therefore, is that an endogenous agent, probably allergy in some form

is the causative factor in those cases in which no other constitutional factor, such as anemia, leukemia and renal disorders, can be found and that the idea that external trauma produces the bleeding must be discarded. GROVE, Milwaukee.

Miscellaneous

COLD VACCINES: AN EVALUATION BASED ON A CONTROLLED STUDY. H. S. DIEHL, A. B. BAKER and D. W. COWAN, J. A. M. A. **111**:1168 (Sept. 24) 1938.

Diehl and his coworkers conducted a three year study of cold vaccines on students at the University of Minnesota. The students were selected so as to exclude those with allergic rhinitis or chronic sinal infection, which might alter the findings in the study. The subjects were divided into experimental and control groups, the former receiving vaccine and the latter receiving a placebo. None of the students knew whether he received the vaccine or the placebo, and the physicians assigned to the health service did not know to which group any of the students belonged.

The colds were duly reported, and a careful tabulation was kept of the time lost from school. These reports were collected quarterly and were checked by the conferences which the doctors in charge of the study had with the students. The vaccine selected for this study was standardized according to the milligrams of nitrogen per cubic centimeter, as follows: pneumococci 0.015 mg., streptococci 0.015 mg., Bacillus influenzae 0.01 mg., Micrococcus catarrhalis 0.0075 mg. and staphylococci 0.0075 mg. It was prepared by the so-called Kreuger method; that is, the organisms were destroyed mechanically instead of by heat. This method has all the merits of vaccines with heat-killed organisms and possibly more. The dose for the experimental group was 0.5 cc. twice weekly for three weeks and then 0.5 cc. every two weeks throughout the fall, winter and spring. The control group received the same dosage at the same intervals, but the solution administered was physiologic solution of sodium chloride. Oral administration of polyvalent vaccine was given to another experimental group, and capsules of lactose were used as a control. Careful records were kept by the students and the doctors assigned to the health service regarding the incidence of colds, the well-being of the student and other factors.

Dr. E. C. Rosenow supplied the authorities with a streptococcus cold vaccine prepared for use by mouth. The streptococci in the nasopharynx or sputums of patients suffering from the common cold furnished this material. Lactose-filled capsules were used by the control group.

The conclusions arrived at may be summarized as follows: The group receiving vaccine subcutaneously experienced an average of 25 per cent fewer colds per person than did the control group. This difference occurred during both years of study and is statistically significant. Practically, however, it is of little or no importance, because a reduction of 25 per cent in the average number of colds in a group of persons is not sufficiently great to justify the time and expense involved in carrying out the intensive vaccination procedure which was utilized. The group which received the polyvalent vaccine administered orally experienced just as many colds as the control group during both years of study. The results reported by the students who took Rosenow's streptococcus vaccine parallel exactly those reported by the control group. Although the data are not entirely conclusive, there is no evidence in this study either that vaccines reduce the complications of colds or that the condition of the nose and throat is related to the frequency of colds in the cold-susceptible group. GORDON, Philadelphia.

ASEPTIC PURULENT MENINGITIS FOLLOWING INJECTION OF ALCOHOL INTO THE FORAMEN OVALE. R. LAFON, Rev. de laryng. **59**:604 (June) 1938.

A patient had facial neuralgia which originated in the teeth and maxillary sinus. Repeated puncture of the sinus failed to relieve the condition, and the injection of

alcohol into the foramen ovale was used. Seven days after the injection of 2 cc. of alcohol the patient showed meningeal symptoms. The spinal fluid was sterile, but 300 polymorphonuclear cells were present. One week later the meningeal symptoms had subsided, but the patient still showed considerable lassitude. A final lumbar puncture aided in the complete recovery. The meningitis was apparently due to the irritating effect of the alcohol on the tissues. The neuralgic pains were relieved from the time of the injection.

BATSON, Philadelphia.

RARE MORBID RELATIONS IN DISEASES OF THE NOSE AND THROAT. H. RICHTER, Monatschr. f. Ohrenh. **72**:1067 (Nov.) 1938.

This contribution represents a collection of cases which the author considers curiosities or rarities. The report includes the following cases: (1) collateral edema of the orbits in acute ethmoiditis, (2) emphysema of the eyelids due to blowing of the nose, (3) carcinoma of the paranasal sinuses affecting both sides within nine years, (4) tumor of the cervical glands due to metastatic extension of an epipharyngeal tumor, (5) chronic laryngitis with pachyderma in a patient for whom the erroneous diagnosis of carcinoma of the vocal cords was made, (6) posticus paresis of the right cord and recurrent paresis on the left side following influenza (the motility of the vocal cords became normal after one month), (7) suspected foreign body in a 5 year old boy, which proved to be acute exudative tracheobronchitis, (8) phlegmon of the larynx in a woman of 66 probably following an acute exacerbation of chronic tonsillitis (the phlegmon was accompanied by fixation of both vocal cords; recovery followed conservative treatment), (9) prelaryngeal thyroid gland containing a cyst filled with colloid material and (10) recurrent paralysis due to a metastasis in the mediastinum from a carcinoma of the pancreas.

LEDERER, Chicago.

AMAUROSIS FOLLOWING INJECTION OF THORIUM DIOXIDE INTO THE MUCOUS MEMBRANE OF THE SEPTUM. JOSEPH BERBERICH, Pract. oto-rhino-laryng. **1**:206 (July) 1938.

The author reports a case of amaurosis with paralysis of the muscles of the eye, fixation of the pupil and interference with the blood supply to the retina following injection of a colloidal suspension of thorium dioxide into the mucous membrane of the septum. The entire picture resembled that of spasm or of occlusion of the central artery of the retina.

The injection was made in a case of ozena. The first injection, into the entire mucous membrane of the septum and the floor of the nose, did not produce an untoward reaction. On subsequent examination it was noted that the mucous membrane of the posterior portion of the septum had not been infiltrated, and consequently a second injection was made posteriorly in the vicinity of the posterior ethmoid artery. There was an immediate reaction, and the patient complained of pain in the eye, blindness and inability to move the eye.

The author believes that, possibly because of anastomosis between the posterior ethmoid artery and the ophthalmic artery, some of the thorium dioxide was forced into the central artery of the retina. The amaurosis was permanent, because thorium dioxide is nonabsorbable, although there was slight improvement in the paralysis of the upper lid.

Four other cases are cited from the literature, but in these cases, as the condition followed injection of an absorbable solution, usually procaine hydrochloride, the amaurosis was transient.

PERSKY, Philadelphia.

WHAT MUST THE OTORHINOLARYNGOLOGIST KNOW CONCERNING THE TREATMENT OF THE SPEECH AND THE VOICE? M. NADOLECZNY, Ztschr. f. Hals-, Nasen- u. Ohrenh. **44**:1, 1938.

In an extensive discussion, the author makes a detailed analysis of disturbances both functional and organic that involve both the voice and the speech. He

describes various diagnostic procedures to differentiate functional from organic disturbances. He describes also the method of production of the voice, with a detailed analysis of the various ranges of tone found in infants, in children of preschool and school ages, in persons undergoing sexual maturity and in adults both male and female. He also differentiates the range of tone in the voice as used in ordinary conversation, in public speaking and in singing. He places a great deal of emphasis on the limitation of the voice within a definite register of the tone scale. In discussing disturbances of the voice during growth, he mentions the various factors that may influence (*a*) retention of the child's voice (eunuchoid), (*b*) change of the falsetto voice and (*c*) interference in the mutation of the voice.

He then discusses in detail the various disturbances of the voice in public speakers, orators and singers, stressing the faulty use of the voice, particularly its extension beyond the person's register, faulty technic in speaking and in singing and acute or chronic straining during the usual occupation, particularly during a local or general illness. He stresses also the treatment of these disturbances.

Discussing the symptoms and sources of disturbances of the voice in orators, singers and others, the author says that organic disorders impair the function only slightly as a rule. A good tenor can sing the part of Lohengrin when he has tonsillitis. It is important to overcome the belief that the sensations of discomfort have organic sources. Such sensations are pressure, dryness, itching, burning, scratching, imaginary presence of a foreign body and even pains in the throat. The patients localize the sensations in various regions, and in the course of a careful examination it is usually possible to differentiate true and functional discomfort. These sensations may remain after catarrh. They occur also in many disorders, such as hyperthyroidism, chronic nephritis, spastic obstipation, gastrointestinal disease and dysmenorrhea. Finally, sensations of discomfort and even pain in the throat may be psychogenic, induced by disorders of the throat in the environment (tuberculosis, cancer) or resulting from psychic shock.

In the discussion of the diagnosis of these conditions, the author stresses, first, a most careful history, with emphasis on causative factors and psychoneurotic conditions, and then careful examination of the larynx. He mentions also a considerable number of erroneous differential diagnostic conclusions and erroneous methods of treatment for each individual condition. He advocates a considerable amount of patience in the correction of these vocal defects. Vocal exercises are perhaps the basis for the correction of many of the disturbances. He advocates both vocal exercises and psychotherapy in the treatment of hysterical and psychoneurotic hoarseness. Organic lesions in the larynx must be treated as indicated, but in order to obtain the most beneficial results vocal exercises and training must be added to the usual repertory of treatment.

In the discussion of disturbances of speech, he describes in great detail the development of speech with its abnormalities, the problem of speech in the deaf and dumb, stammering and its causes, lisping, nasal speech, due probably to an occlusion of either the nares or the nasopharynx, and stuttering.

In conclusion, he states that treatment of disorders of the speech and the voice must be considered from the biologic, from the psychic and psychoneurotic, from the functional and from the organic standpoint, and that successful results can be obtained only by careful analysis of the foregoing factors and application of suitable therapy in each individual case.

PERSKY, Philadelphia.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

THOMAS W. LEWIS, M.D., *President*

Regular Monthly Meeting, Dec. 5, 1938

SCLEROMA (RHINOSCLEROMA): HISTOLOGIC CHANGES AFTER TELERADIUM THERAPY; REVIEW OF SCLEROMA IN THE UNITED STATES. Presented by DR. J. A. WEISS.

This paper will be published in full, with discussion, in a later issue of the ARCHIVES.

AIR CELLS IN THE PETROUS PART OF THE TEMPORAL BONE OF A CHILD OF THIRTEEN. Presented by DR. J. GORDON WILSON.

I shall present an anatomic study of air cells in the petrous part of the temporal bone of a child aged 13 and of their relation to the middle ear, discussing the petrous part as a whole, which, because of its part in the formation of the cranium and its contents, I regard as the most interesting bone in the skull.

There are differences in the extent, size and location of the tubal cells in different persons. The specimen to be discussed was chosen because it presented well marked air cells and other anatomic features which are believed to be typical. Examination shows that infection not only may come from the general vascular system but may spread from the middle ear. Difficulty arises in studying the structure of the temporal bone and in differentiating between air cells and marrow cells, and the method of preparing my specimens may be outlined.

In summary it may be be stated: 1. Air cells in the petrosa were well developed in this 13 year old child. 2. They came from two independent sources: (*a*) as extension of the mastoid cells and (*b*) from the hypotympanic space of the middle ear. They do not communicate with each other. 3. The tubal cells lie chiefly in the inferior part of the petrous arc and are related to the carotid canal. They open into the hypotympanic space by a distinct channel or opening.

DISCUSSION

DR. BARRY J. ANSON: I wish to comment on Dr. Wilson's paper by presenting for comparison the anatomy of the apical cells in a younger child, aged 3½ years. I shall demonstrate with lantern slides the topography and shape of the air cells at the petrous tip, selecting four illustrative levels. In this specimen the tympanic orifice is similar in form and location, but distributed more than in the preceding one. My associates and I have wax plate reconstructions prepared from the serial sections of this specimen.

Section 1 is taken through the cranial opening of the carotid canal and the upper portion of the cochlea and of the superior semicircular canal. The cells are near, but not continuous with, the epitympanic and mastoid cells which surround the superior semicircular canal; they do not reach the internal acoustic meatus.

Section 2 is taken through the vestibular (oval) window, the ossicles and the auditory tube. The cells at this horizontal level are at their maximal extent; as yet they are without communication with the tympanic cavity (middle ear); they pass in an interconnected group beneath the carotid canal to the cranial side of the petrous tip, where they are separated from the dura mater by a thin plate of bone (inner table).

Section 3 is taken near the floor of the auditory tube, through the tympanic membrane, the attached malleus and the fossa for the jugular vein. The apical air cells open, by broadened communication (tympanic orifice or ostium), into the space of the middle ear.

Section 4 is taken at the floor of the tympanic cavity, near the inferior attachment of the tympanic (drum) membrane. Here, close to their inferior limit, the air cells still open into the lowermost part of the tympanic cavity (middle ear) by several additional communications (comparable to nasal orifices or ethmoid cells). They lie in the walls of the canal for the carotid artery and the fossa for the jugular vein.

Reconstructions.—As verified graphically in reconstructions of the air cells and associated structures, the cells extend upward and arch over the carotid canal to reach its cranial side; anteriorly they extend toward the cochlea, while posteriorly they pass along the carotid canal and then beneath the latter; they communicate with the tympanic cavity by a definite orifice or ostium situated at the horizontal level of the vestibular window; they communicate additionally by openings situated on the floor of the cavity, near the opening of the auditory tube. The two large groups of cells (passing over and beneath the carotid canal) approach each other, yet do not actually meet, on the cranial side of the canal; they surround the carotid canal almost completely. In general, the cells resemble those of the mastoid portion of the temporal bone. The greater complexity in form of the apical cell group is seemingly due to the presence of the carotid canal; encountering this obstacle in the course of their invasion of bone they pass along, under and above it, pneumatizing its bony wall. The anatomy is fully described in a previous publication (Anson, J. B.; Wilson, J. G., and Gaardsmoe, J. P.: Air Cells of Petrous Portion of Temporal Bone in a Child Four and a Half Years Old, ARCH. OTOLARYNG. **27**:588 [May] 1938).

DR. S. A. SCIARRETTA: I wish to call attention to another work on this subject written by Federici of the University of Genova (Federici, F.: Petrosite anteriore o bullite? A proposito di un caso di cosi detta petrosite anteriore, *Arch. ital. di otol.* **48**:413 [July] 1936). He reported an interesting case, in which several operations produced little improvement. Finally, when the peritubal air cells were discovered to be infected and were opened, the patient made an uneventful recovery. In his review of the literature, the author pointed out the numerous classifications advanced by many observers. He concluded that the various groups of air cells found in the temporal bone should be separated into two distinct divisions.

The cells located anteriorly and inferiorly to the petrosal pyramid should be called the bullar air cells; in this group he included the subcochlear, peritubal and pericarotid air cells. I think this is the group of air cells which Dr. Wilson has so masterfully described and illustrated this evening. Federici stated that pathologic processes invading this group of cells invariably originate from the middle ear.

The second group of air cells comprises those which originate from the antrum and are located posterosuperiorly and posteroinferiorly to the pyramid, that is, the sublabyrinthine, supralabyrinthine and translabyrinthine air cells.

DR. ALFRED LEWY: I should like to ask Dr. Wilson or Dr. Anson if we are to understand from their presentation that there are only two origins for cells which appear in the petrous pyramid, one group communicating with the tympanic cavity in the region of the eustachian tube and the other with the epitympanic space. It has been generally held by clinicians that there are groups of cells with other origins in the petrosa. I should like to ask whether the cells near the internal auditory meatus which can be reached from the posterior fossa are part of either of these groups or whether they constitute a separate group. If I understood Dr. Wilson correctly, he referred to the cells that surround the labyrinth as belonging to the epitympanic group.

DR. ELMER W. HAGENS: I have enjoyed this presentation by Dr. Wilson and Dr. Anson. There is one point that I should like Dr. Wilson to bring out. He showed a thickened tunica propria in the pneumatic space. I have a specimen

from a child aged 3 years with a pneumatic space in the petrous apex, in which the tunica propria is thick. I was puzzled about it and explained it by saying that the tunica propria was thickened because the epithelium had not yet shrunk back and down to the bone, but Dr. Wilson's specimen was from a child of 13. If these pneumatic spaces develop early, by the fifth year even, and if the development started at that age in his case, it must then take a long time for them to shrink. In adults it is known that the epithelium is down practically to the bone. It is interesting to find a child of 13 with pneumatic spaces developing and with the tunica propria so thick. Surely, at certain levels one sees no lumen at all.

I have not had occasion to examine a child with pneumatic cells developed at that age with such a thickened tunica propria. The ears of patients presenting that condition had infection, and it was explained on that basis. I should like to know whether Dr. Wilson has any idea of the length of time it takes for shrinkage of the tunica propria from childhood to the adult stage.

Dr. J. Gordon Wilson: In reply to Dr. Lewy, in the petrous portion of the temporal bone there are two groups of cells with separate origins. Air cells lying along the wall of the tympanic cavity are not in groups similar to those in the mastoid or those I have described in the petrous pyramid.

As Dr. Hagens says, the tunica propria usually is thick at the age of 3 years. In the fetus, the middle ear is filled with embryonic mesenchymatous tissue, which gradually shrinks, leaving clear areas in which the epithelium shows itself. This mesenchyme is replaced by fibrous tissue, which extends to the antrum and helps in forming the air cells. After the first year, development goes on rapidly until the child is about 5 years old, when it slows down. The apparent thickness of the tunica propria in one of my sections is due to the plane of the section. Before the advancing connective tissue and epithelium help to form the air cells, I believe, there is softening of the adjacent bone. Osteoclasts are few and may be wanting. What happens to formation of cells when the potential connective tissue and epithelium entering from the middle ear is altered by disease, either before or after birth, is disputed.

The Structure of the Ethmoid Labyrinth (Anatomic Study). Presented by Dr. O. E. Van Alyea.

This article will appear in full, with discussion, in a later issue of the Archives.

Book Reviews

A Treatise on the Surgical Technique of Otorhinolaryngology. By Georges Portmann and collaborators, translated by Pierre Violé, M.D. Price, $12.50. Pp. 675, with 474 illustrations and 2 colored plates. Baltimore: William Wood & Company, Medical Division, The Williams & Wilkins Co., 1939.

Georges Portmann, Professor of Otorhinolaryngology at the Medical School of the University of Bordeaux, has been well known to American otolaryngologists for years. Most of them have read his articles, heard his presentations on the occasions of his several trips to this country or taken his popular courses in Bordeaux. For this reason his book on the surgical technic of otolaryngology will be studied with much interest and will be a welcome addition to many a physician's library. The work has been admirably translated by Dr. Pierre Violé, the well known Los Angeles specialist, who has had charge of the American edition.

The book is attractively printed on good paper in the popular two column style, which makes for easy reading. There are 675 pages and 474 illustrations. The latter are exceptionally well done drawings, some life size, some larger than life size and a few on a reduced scale, that adequately illustrate each important step of operative technic in the surgical proceeding under description. There are, for instance, 26 cuts illustrating radical mastoidectomy, 17 illustrating the operation on the frontal sinus, 18 illustrating total laryngectomy and a proportionate number illustrating other operations. The text accompanying the illustration is concise and easy to read and understand. It follows a fairly uniform pattern; for instance, in the discussion of radical mastoidectomy the following points are covered: definition, indications, preparation of the patient, anesthesia (the technic of local anesthesia is given in detail in five steps), instruments, lighting, position of the patient, position of the surgeon and his assistant, operative field, operation itself (divided into 13 steps), operative incidents, postoperative care by days and postoperative incidents. Each step is explained in concise detail, which is followed with ease.

All the usual surgical procedures of otolaryngologic practice are described, but since this work is purely one on surgical technic, little space is devoted to symptomatology or to surgical indications, although these are briefly mentioned. Most of the descriptions are those of standard methods, although, of course, somewhat modified according to the preferences of the author as developed by his wide experience. American-taught students will have little difficulty in adjusting the methods taught them with the teachings of Dr. Portmann, since differences are few. This book should be most valuable as a textbook for graduate students of otolaryngology, for residents and for those who wish to "brush up" or to improve their operative technic. Even the mature operator will find in its pages a wealth of interesting and helpful material.

A paragraph from the preface briefly but well describes the work:

"My object has been to present practically but modestly, in nearly telegraphic style, a procedure for each affection without useless phraseology. I desire to give the essentials that one must know from the time preceding the operation until recovery has been definitely established. If I were to express myself thus, I would have wished that the book be a 'spoken' one, a repetition of that which my students hear me say each day in the operating room."

Dr. Portmann, his collaborators and Dr. Violé are to be congratulated on the production of a most attractive and useful book.

Directory of Otolaryngologic Societies *

FOREIGN

Collegium Oto-Rhino-Laryngologicum Amicitiæ Sacrum

President: Dr. Louis Ledoux, Brussels, Belgium.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg 143, Groningen, Netherlands.
Place: Brussels, Belgium. Time: September 1939.

Hungarian Otolaryngological Society

President: Dr. J. Treer, Bocskay-u. 9, Szeged.
Secretary: Dr. G. Kelemen, Reáltanoda-u. 9, Budapest.

Sociedad Rioplatense de Oto-Rhino-Laringología (Argentine Section)

President: Dr. Raul Becco, B. Mitre 1690, Buenos Aires.
Secretary: Dr. Juan Manuel Tato, Santa Fé 1171, Buenos Aires.

Société Française d'Oto-Rhino-Laryngologie

Secretary: Dr. Henri Flurin, 19 Avenue Mac-Mahon, Paris, 17e.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. Leroy A. Schall, 270 Commonwealth Ave., Boston.
Place: St. Louis. Time: May 15-19, 1939.

American Academy of Ophthalmology and Otolaryngology

President: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 8-13, 1939.

American Bronchoscopic Society

President: Dr. John D. Kernan Jr., 120 E. 75th St., New York.
Secretary: Dr. Lyman Richards, 319 Longwood Ave., Boston.
Place: Westchester Country Club, Rye, N. Y. Time: May 27, 1939.

American Laryngological Association

President: Dr. George B. Wood, 504 Physicians' Bldg., Philadelphia.
Secretary: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Place: Westchester Country Club, Rye, N. Y. Time: May 24-26, 1939.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Harold I. Lillie, Mayo Clinic, Rochester, Minn.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Drake Hotel, Chicago. Time: May 9-11, 1939.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

SECTIONS:

Eastern.—Chairman: Dr. Frank E. Kittredge, Masonic Temple, Nashua, N. H.
Southern.—Chairman: Dr. Francis E. LeJeune, Maison Blanche Bldg., New Orleans.
Middle.—Chairman: Dr. T. R. Gittins, Davidson Bldg., Sioux City, Iowa.
Western.—Chairman: Dr. Frederic G. Sprowl, Medical Arts Bldg., Spokane, Wash.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Isidore Friesner, 36 E. 73d St., New York.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.
Place: Westchester Country Club, Rye, N. Y.: Time: May 22-23, 1939.